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DELAYED OPERATION IN THE TREATMENT OF THE PERFORATED APPENDIX

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DELAY in operation upon a purely surgical intra-abdominal condition does not appeal, at first sight, to surgical instincts. Such practice in the care of the perforated appendix has never become universally popular in the forty-six years since it was first advocated by A. J. Ochsner[1] and at present it continues to represent one of the most controversial questions of surgical practice.[2]

We were trained in the belief that the only treatment for the perforated appendix was immediate operation, and such was our practice when the Duke Hospital opened in July, 1930. It soon became evident, however, that in some cases immediate operation did more harm than good. Convinced that the principles of delayed operation were surgically and physiologically sound, and encouraged by the statistics of those who had used this form of treatment, we gradually came to adopt the plan of delayed operation in all patients in whom a definite preoperative diagnosis of perforated appendicitis could be made. This study records our experience with 248 cases of appendicitis with perforation admitted to the surgical service of the Duke Hospital from July, 1930, to July, 1937 (Table I).

The rationale of delaying operation in cases of appendiceal peritonitis is based upon the fact that a natural protecting mechanism exists within the abdomen which, if left undisturbed, is usually capable of causing the resolution or localization of appendiceal peritonitis. This protective mechanism consists of the inflammatory response to the presence of irritation within the peritoneum and is characterized by the outpouring of an exudate which is bactericidal and which contains leucocytes and fibrin. The fibrin agglutinates peritoneal surfaces of bowel, omentum, and abdominal wall in an effort to wall-off areas of inflammation from uncontaminated parts of the peritoneal cavity. This being possible, the

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TABLE I

GENERAL ANALYSIS OF ALL CASES STUDIED

1930-1937	IMMEDIATE OPERATION		DELAYED OPERATION*		SUBSIDED NO OPERATION†		TOTAL	
	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	DEATHS
Acute appendicitis	685	0	0	0	0	0	685	0
Local peritonitis	65	5	2	0	13	1	80	6
Abscess	75	7	10	3	50	2	135	12
Diffuse peritonitis	31	14	1	0	1	1	33	15
Total	856	26	13	3	64	4	933	33

*The conservative treatment was abandoned after an average period of 9 days because signs were not subsiding or were increasing.

†Symptoms and signs subsided completely or the patient died under conservative treatment without operation. Surviving patients discharged after an average hospitalization of 16 days to return for appendectomy in 3 months.

bactericidal effect of the inflammatory reaction is sufficient to cause complete resolution of the infection in the majority of properly managed cases. Such a protective mechanism is effective only if left undisturbed, disturbing elements being any factor, such as food or cathartics, which stimulates peristaltic activity in the bowel, or any mechanical procedure, such as operation, either of which may break up the protective barrier of agglutinated peritoneal surfaces and open the uncontaminated peritoneum to infection.

This concept was first applied by Ochsner in the preoperative treatment of diffuse appendiceal peritonitis. In this condition he advocated complete rest of the bowel by withholding everything by mouth, gastric lavage if the stomach and intestinal tract were not empty, and the administration of parenteral fluids until the peritonitis had localized or subsided when operation could be done safely. In his hands, in those of Deaver,[3] who soon became a staunch advocate of this plan of treatment, and more recently in the hands of Guerry[4] striking reductions in the mortality of diffuse peritonitis of appendiceal origin were demonstrated.

Nevertheless, although almost half a century has elapsed since it was first advocated, delayed operation has never been adopted by the majority of surgeons. Why has it not been accepted more uniformly? Royster[5] says that "the Ochsner treatment has been woefully misunderstood" and "dissatisfaction with its results or fatal consequences following its use have come about either through inability to comprehend its principles or to careless execution in its technique." Such misunderstanding may be caused by the fact that Ochsner's descriptions of his plan are not easily accessible and that his followers in this country have not described their technique in detail. Moreover, set rules governing the operation of the plan are difficult to outline, for its successful operation requires judgment which is intangible and does not easily lend itself to a list of didactic rules which may be easily followed. Probably a

more important cause for the lack of popularity of the treatment is the fact that it is more difficult to manage than immediate operation. It necessitates care in selecting cases, close supervision during the course of treatment, and keen judgement in determining when operation should be done once the treatment is started. It requires the sympathetic understanding of the family who usually feel that their procrastination in bringing the patient late may be compensated for by immediate operation, and of the family doctor who may not understand the treatment. And, in addition, it requires the cooperation of the patient who, in the face of continued improvement in his condition, must take nothing by mouth for a longer period of time than he finds comfortable. Such difficulties, we believe, however, are far outweighed by the shorter period of hospitalization, decrease in incidence of complications, and lowered death rate which accompany this form of treatment.

SELECTION OF CASES

Stage of Disease.—There should be no misunderstanding that delayed operation is reserved only for patients with perforated appendices. Whether or not the appendix has perforated in any given case must be determined individually for each patient after a careful analysis of history and physical and laboratory findings. No definite hour after onset of symptoms can be used as a criterion of perforation. While it is true that in most severe attacks of appendicitis the appendix is found to be ruptured forty-eight hours or longer after an attack, it is not wise to set an arbitrary hour, such as forty-eight hours after onset, as a dividing line to separate cases requiring immediate or delayed operation. We have seen one patient in whom delayed operation was justified in the first day of the illness and have seen many in whom immediate operation was indicated four or five days after onset of symptoms. It must be emphasized that, if after careful evaluation of all diagnostic measures there exists a reasonable question as to whether the appendix is or is not ruptured, immediate operation must be done. We have followed this rule rigidly and have found that in our group of 171 cases with perforated appendices in which immediate operation was done 96, or 56 per cent, were in patients with symptoms of forty-eight hours' duration or less. (Table II.)

Differences of opinion exist as to which forms of appendiceal peritonitis should be treated conservatively. Ochsner first advocated his treatment in cases of spreading or diffuse peritonitis. In England, where Sherren[6] first advocated delayed operation in 1905, it has been used largely in cases where localization has already occurred, while patients with diffuse peritonitis, unless moribund, are usually submitted to immediate operation. British surgeons also frequently treat inflammatory masses conservatively for a long period of time. In the United States

TABLE II

DURATION OF SYMPTOMS ACCORDING TO TREATMENT SELECTED—ALL CASES OF PERFORATED APPENDICITIS

DURATION OF SYMPTOMS	IMMEDIATE OPERATION		NO OPERATION		UNSUCCESSFULLY DELAYED		TOTAL	
	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	MORTALITY %
1 to 24 hr.	19	0	1	1*	1	0	21	4.8
25 to 48 hr.	72	10	3	0	0	0	75	13.3
49 to 72 hr.	28	3	11	0	1	0	40	7.5
4 days	18	4	8	2†	4	0	30	20.0
5 days	9	4	8	0	0	0	17	23.5
6 days	7	3	10	0	1	0	18	16.7
7 days	2	0	4	0	2	0	8	0
8 days	1	0	1	0	0	0	2	0
9 days	3	0	2	0	1	0	6	0
10 days	2	1	5	0	1	1§	8	25
Longer	10	1	11	1‡	2	2¶	23	17.4
Total	171	15.2%	64	6.2%	13	23%	248	13.3%

*Death from general peritonitis.
†Death from pneumonia.
‡Death from retroperitoneal abscess.
§Death from subphrenic abscess.
¶Death from pulmonary embolus.

conservative treatment has been reserved largely for patients with diffuse or spreading peritonitis, and, when inflammatory masses have appeared, early operation has usually been advocated. We have combined these two applications of the conservative regime and now treat all patients with ruptured appendices conservatively, with the exceptions as listed below, regardless of the stage of the peritoneal infection in which the patient is first seen. Our experience (Table I) has been largest in patients with inflammatory masses present on admission. Only two patients with diffuse peritonitis have been treated conservatively. In one the infection localized satisfactorily and was drained. The other patient entered the hospital in the twenty-fifth hour of her illness with signs of generalized peritonitis, maximum in the pelvis, thought to be coming from a salpingitis. Placed on the conservative regime, she seemed to improve, although her temperature and pulse did not fall, and she died on the fifth day of generalized peritonitis. This case, early in our experience, corroborates the observations of Alton Ochsner[7] and has convinced us that expectant treatment is unwise in the presence of early perforation of the appendix and widespread signs of peritonitis. Such a situation, we now feel, represents the only major contraindication to the use of delayed operation.

Age of Patient.—A. J. Ochsner recognized early in his experience that delayed operation was not borne well by the very young or the very old. Aged patients, he emphasized, do not stand confinement to bed well, while in small children the omentum is not sufficiently developed to act as a protection. This opinion has been modified by Alton Ochsner[7]

who recommends the conservative regime in children in whom localization of the peritoneal infection is beginning. And more recently it has been reversed by the experience of Coller and Potter[8] at the University of Michigan and Adams and Bancroft[9] at the University of Minnesota who have found the use of delayed operation advisable in children with diffuse as well as localized appendiceal peritonitis.

Coller and Potter[8] in 1934 reported a series of 48 children with diffuse peritonitis treated conservatively with a mortality of 12.5 per cent. Although recognizing that the delayed form of operative treatment is less efficient in children than in adults, as is any form of treatment in this age group, their results using delayed operation were superior to immediate operation and they recommend the use of the conservative regime in all patients with diffuse peritonitis regardless of age. Adams and Bancroft[9] in 1937 reported 110 children with appendiceal peritonitis treated conservatively. Sixty-seven of these patients had generalized peritonitis. Sixty-one per cent developed inflammatory masses, only 10 per cent of which formed abscesses which had to be drained. Ninety-five of the remaining cases subsided completely and were discharged to return for interval appendectomy in eight weeks. The mortality in this series of 110 cases was 4.5 per cent.

Our practice has been to treat children with appendiceal peritonitis conservatively only when localization is already beginning, and to use immediate operation for children with diffuse peritonitis. We have used the conservative treatment in 15 children (Table III). In 11 of these, symptoms and signs subsided entirely and the patients left the

TABLE III

AGE DISTRIBUTION ACCORDING TO TREATMENT SELECTED—ALL CASES OF PERFORATED APPENDICITIS

AGE	IMMEDIATE OPERATION		NO OPERATION		UNSUCCESSFULLY DELAYED		TOTAL	
	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	MORTALITY %
1 to 5 yr.	8	5	1	0	0	0	9	55.5%
6 to 10 yr.	22	4	3	0	2	1§	27	18.5%
11 to 15 yr.	35	5	7	0	2	0	44	11.4%
16 to 20 yr.	28	1	13	1*	1	0	42	4.8%
21 to 30 yr.	35	4	12	0	2	0	49	8.2%
31 to 40 yr.	16	2	12	0	0	0	28	7.2%
41 to 50 yr.	15	2	6	1†	4	1¶	25	16.0%
51 to 60 yr.	11	3	6	2‡	1	0	18	27.7%
61 to 70 yr.	1	0	2	0	1	1¶	4	25%
71 to 80 yr.	0	0	2	0	0	0	2	0%
Total	171	15.2%	64	6.2%	13	23%	248	13.3%

*Death from generalized peritonitis.
†Death from retroperitoneal abscess.
‡Death from pneumonia.
§Death from subphrenic abscess with rupture through diaphragm.
¶Death from pulmonary embolus.

hospital without operation, to return in three months for interval appendectomy. The remaining 4 failed to improve and were operated upon on an average of eight days after admission. One died of a subphrenic abscess which ruptured through the diaphragm. All the others recovered.

In children with diffuse peritonitis, it has been our practice to use immediate operation. Jones and Menefee,[10] in an analysis of all cases of appendicitis in our children's ward between 1930 and 1935, found our mortality in 12 children with generalized peritonitis, all treated by immediate operation, to be 42 per cent. In the past three years 11 children with generalized peritonitis have been treated, 10 by immediate operation and 1 by delayed operation. Five of those subjected to immediate operation died. We are not satisfied with this result which contributes largely to our mortality of 42.8 per cent in all cases of diffuse peritonitis treated in the past three years (Table IV). And in the future we intend to follow the practice of Coller and Potter and Adams and Bancroft and treat children with generalized appendiceal peritonitis conservatively the same as adults.

TABLE IV

ANALYSIS OF ALL CASES OF PERFORATED APPENDICITIS SHOWING THE IMPROVEMENT IN MORTALITY RATE IN THE PERIOD 1934-1937 WITH CONSISTENT USE OF THE CONSERVATIVE REGIME WHEN INDICATED EXCEPT IN CASES OF DIFFUSE PERITONITIS IN CHILDREN

1930-1934	LOCALIZED PERITONITIS		ABSCESS		DIFFUSE PERITONITIS		TOTAL	
	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	DEATHS	NO. CASES	DEATHS
Immediate operation	34	3	45	5	18	8	97	16
Unsuccessfully delayed	0	0	7	3	0	0	7	3
No operation	6	1	11	1	1	1	18	3
Total	40	10%	63	14.3%	19	47.4%	122	18%
1934-1937								
Immediate operation	31	2	30	2	13*	6	74	10
Unsuccessfully delayed	2	0	3	0	1	0	6	0
No operation	7	0	39	1	0	0	46	1
Total	40	5%	72	4.2%	14	42.8%	126	8.7%

*Ten of these were in children under 13 years of age, of whom 5 died. The remaining 3 were in adults with symptoms of less than two days. One of these died the day of operation of shock.

Previous Catharsis.—The recent ingestion of cathartics has been stated by Bailey[11] to be a justifiable reason for not using the delayed treatment. It is our feeling that such catharsis represents a hazard equally as dangerous if immediate operation were undertaken and we have rarely been influenced by a history of catharsis during the attack. Of our 77 cases treated conservatively, 37 had had catharsis.

Questions of Diagnosis.—When a definite differential diagnosis between perforated appendicitis and some other intra-abdominal catastrophe which requires immediate operation cannot be made, immediate operation should be done. If, on the other hand, the alternative condition suspected does not require immediate operation, indication for delayed operation is all the more certain.

Pregnancy.—Coller and Potter[8] urge that the delayed form of treatment never be used in pregnant women, stating that the presence of the enlarged uterus prevents localization of the infection. Bailey,[11] on the other hand, states that the presence of a pregnancy should not influence one in the selection of suitable cases for delayed operation. We have had no experience with this condition.

Facilities for Care.—It must be emphasized that the conservative treatment of appendiceal peritonitis is not a substitute for operation but a preparation for it. It is not to be undertaken in the home or by the family doctor but in a hospital, "on the threshold of the operating room"[11] under the constant eye of the surgeon who is to operate at any hour if necessary. Adequate care can be given satisfactorily in any modern hospital with a trained nursing staff. However, if the surgeon is not so situated that he can visit his patients personally at least three times daily and be prepared to respond at any hour if called, the delayed form of treatment should not be undertaken.

MANAGEMENT OF CONSERVATIVE TREATMENT

Once a patient is selected for the delayed operative treatment, the following regime is outlined:

Fowler's Position.—The patient is kept in bed in Fowler's position with the head of the bed elevated on six-inch blocks.

Nothing by Mouth.—Although Sherren[6] and his followers in England allow water by mouth, we are convinced of the wisdom of Ochsner's original plan and give absolutely nothing by mouth during the early days of the treatment.

Parenteral Fluids.—A fluid intake of 2,000 to 4,000 c.c. is maintained daily by divided intravenous injections, subcutaneous infusion, or continuous intravenous infusions. Fluids are not administered by rectum nor are large enemas given. Care must be taken that the electrolytes of the blood are not depleted, particularly if constant siphonage of the stomach is maintained.

Gastric Lavage.—If the patient has been vomiting, has eaten recently, or if he is distended, the stomach is washed. If duodenal content is recovered, continuous siphon drainage of the stomach is maintained, using the method of Wangensteen until such drainage stops.

Sedation.—It has been our experience that opiates are seldom necessary. The occurrence of pain during treatment usually indicates that

an abscess should be drained or that the intestinal tract is overloaded and stomach siphonage is necessary. Opiates are used only to allay restlessness or apprehension. We have not used large doses of morphine to increase intestinal tone.

Special Observation.—As soon as the patient is admitted, the extent of abdominal signs is carefully recorded on his chart. If he enters with a palpable inflammatory mass, its exact size is drawn on the abdominal wall with gentian violet. In addition, a special bedside nursing chart is kept on which the nurses record the rectal temperature every two hours, the pulse every hour, and the occurrence of vomiting and pain, as well as the fluid intake, nature and amount of stomach drainage, if any, the urinary output, and the passage of flatus or feces.

CLINICAL COURSE UNDER CONSERVATIVE TREATMENT

There are several possible courses which may be followed by the peritoneal infection under conservative treatment (Fig. 1). They are:

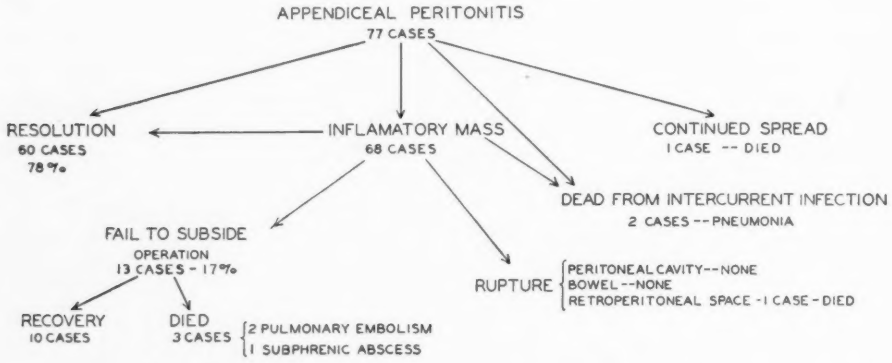

Fig. 1.—Diagram showing the course followed and the result in 77 cases of appendiceal peritonitis treated conservatively. The 77 cases included 2 with diffuse peritonitis, 15 with local peritonitis, and 60 with palpable masses. Of the 60 cases in which signs and symptoms completely resolved and the patients were discharged to return for interval appendectomy, 48 entered with palpable masses and 12 with local peritonitis. Of these 12 cases with localized peritonitis, 5 developed palpable masses which subsided and 7 subsided without first forming a mass. Of the 13 cases which failed to subside and in which operation became necessary, 1 entered with diffuse peritonitis, 2 with local peritonitis, and 10 with palpable masses.

1. The entire inflammatory process may disappear by resolution. This occurred in 60, or 78 per cent, of our 77 cases treated conservatively.

2. The inflammatory process may become localized in the right iliac fossa or in the pelvis, forming a palpable inflammatory mass. Such a mass (a) may then resolve, (b) may fail to resolve or actually increase in size and necessitate drainage, or (c) may rupture into the peritoneal cavity, the bowel, or the retroperitoneal tissues.

3. The inflammatory process may continue to spread and the patient may succumb to a generalized peritonitis.

One must be on the alert for the development of any of these possibilities and if indication of continued spread of the peritoneal infec-

tion or of increase in size or rupture of an abscess occurs, immediate operation must be done.

Resolution of the Peritoneal Infection.—In the majority of patients under the conservative regime of treatment resolution of the peritoneal infection occurs and improvement in the general condition is observed from the start. Vomiting and pain stop, dehydration is overcome, abdominal distention is lessened, tenderness and rigidity of the abdominal muscles decrease, and the temperature and pulse gradually fall.

The continuation of abdominal pain, as is contrasted to abdominal soreness, usually indicates that the appendix is not ruptured or that an abscess under tension is present.

With the complete rest given the gastrointestinal tract and with the use of the indwelling nasal catheter in the stomach, when indicated, abdominal distention has never been a troublesome condition.

The temperature usually subsides slowly and in our experience approximated normal in an average of seven days (Chart I). Maintenance of an elevated temperature in itself for a few days need not be considered abnormal.

The pulse usually falls before the temperature (Chart I). A rising pulse is one of the most important single signs of trouble and usually indicates that the delayed treatment should be abandoned.

If the clinical course progresses satisfactorily, the patient is kept on nothing by mouth until the temperature approximates normal, all distention has disappeared, and he is free of discomfort. Water in amounts of 30 c.c. hourly is then started. If this is not accompanied by abdominal discomfort or rise in temperature, broth, ginger ale, and orange albumin are given in amounts not exceeding 30 c.c. per hour on the following day. This is then increased to 60 c.c. on the next day, clear liquids as desired the next day, and soft diet thereafter during the hospital stay. As feedings are started, the nursing chart, symptoms, and abdominal signs must be watched closely for evidence of a flare-up in the inflammatory process. If at any time abdominal discomfort, increase in abdominal signs, or recurrence of tachycardia or fever appears, all fluid intake by mouth must be stopped. If signs do not then subside promptly, operation must be done. If it is necessary to keep a patient on nothing by mouth for longer than four or five days, as is often the case, transfusions of 500 c.c. of citrated blood are given at one- or two-day intervals. None of our patients undergoing conservative treatment have developed nutritional edema, although we occasionally have seen this in postoperative patients with peritonitis, in whom feeding by mouth has been withheld for a long period of time.

Abscess Formation.—The patient may enter the hospital with a palpable inflammatory mass or one may develop while under treatment.

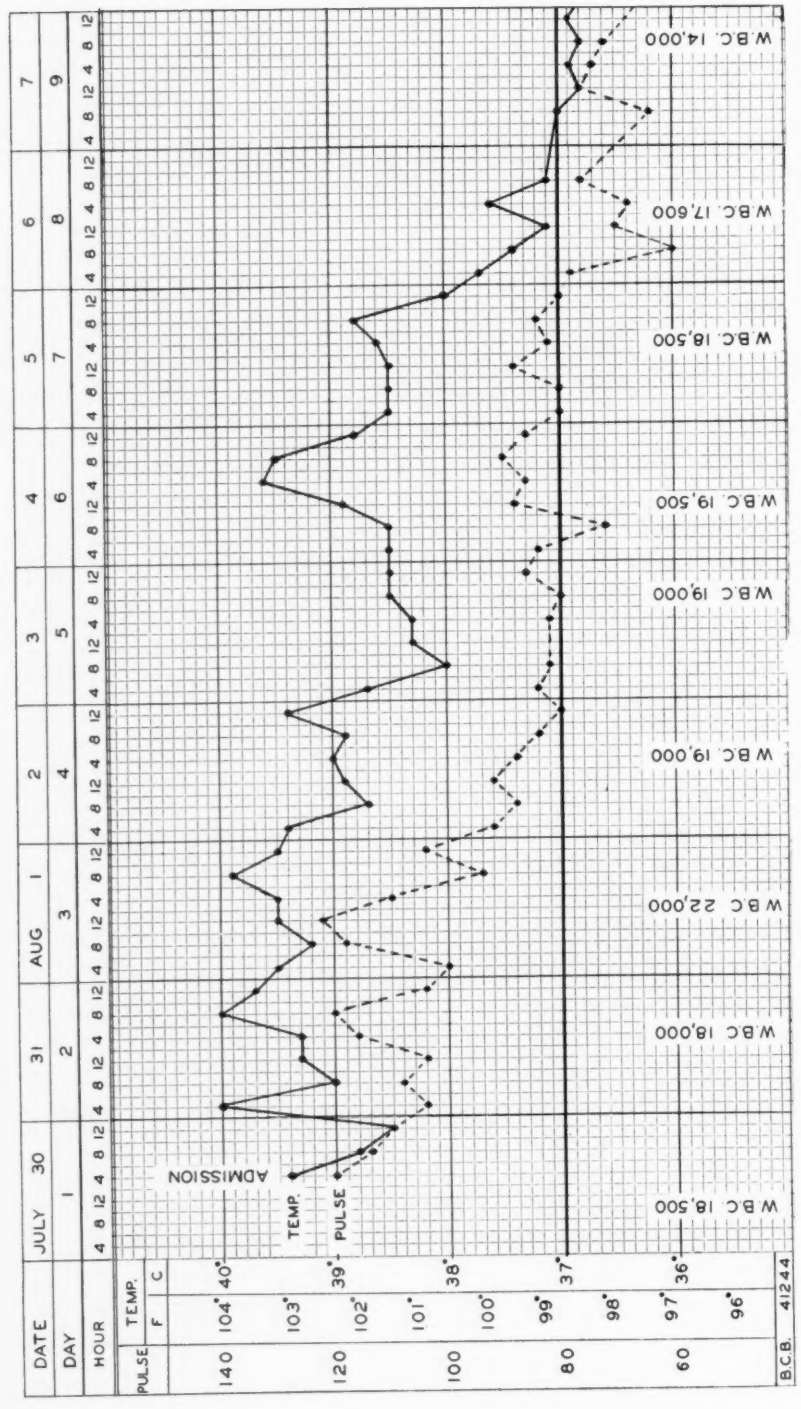

Chart I.—The temperature chart of a typical case with appendiceal peritonitis treated conservatively. The average temperature on admission of 60 cases treated conservatively in whom resolution of the infection occurred was 102° F. and the average pulse 110. The average duration of fever was 7 days. The patient whose chart is shown was a boy of 14 years of age who entered with symptoms of 3 days' duration and with signs of peritonitis in both lower quadrants and in the pelvis. On the second and third days an indefinite mass was found in the right lower quadrant. It could not be felt after the fourth day. Abdominal tenderness had disappeared by the sixth day. Water was given by mouth on the sixth day and clear liquids on the seventh. He remained in the hospital 15 days, the temperature being normal after the eighth day.

It is surprising how often large inflammatory masses will disappear completely under the conservative regime of treatment. Such was the case in 53, or 78 per cent, of the 68 patients who either entered with or developed inflammatory masses while under conservative treatment. In 367 cases of appendiceal peritonitis with inflammatory mass treated expectantly, Sworn and Fitzgibbon[12] report 298, or 81 per cent, to have subsided, 13 by evacuation into the bowel. Love[13] reports 68 per cent of 341 cases and Adams[14] 67 per cent of 214 cases as subsiding completely under the conservative regime.

About 20 per cent of inflammatory masses will not subside and will form abscesses which must be drained. That such an abscess may rupture into the peritoneal cavity must be remembered by all who practice delayed operation. In our series of 68 patients with inflammatory masses on admission or who developed them while under the course of treatment, 13, or 19 per cent, did not subside and were drained at operation, the appendix not being removed in any of these cases. None in our experience have had an abscess rupture into the peritoneal cavity or into the bowel. One patient admitted one month after onset of symptoms died thirty-six hours after admission with a large retroperitoneal abscess.

Close observation of patients with inflammatory masses undergoing the treatment must be maintained so that those with abscesses which need drainage or which might rupture may be selected for operation. Careful evaluation of the temperature, pulse, abdominal symptoms, and size of the abdominal mass must be made frequently. The size of the mass is mapped on the abdominal wall daily and bimanual examination of the pelvis made every two days. The following are taken as indications for operation: (1) The mass is not decreasing in size after four or five days' treatment; (2) the temperature is not subsiding after a week; (3) the mass is growing larger (this is indication for immediate operation); or (4) pain, as contrasted to abdominal soreness, appears. Because of the difficulty in estimating the size of pelvic abscesses, Bailey does not treat abscess in this position expectantly. We feel that bimanual examination of the pelvis can give reliable evidence of the size of pelvic abscesses and have not hesitated to treat abscesses in this position by delayed operation. Coller and Potter draw attention to edema of the genitals as a sign of large intra-abdominal abscesses under tension which require immediate operation. They also feel that a return of elevation in leucocyte count after a preliminary fall during treatment indicates the presence of an abscess.

None of our patients have had an abscess rupture into the peritoneal cavity while under conservative treatment, although Bailey[11] reports such a catastrophe. His patient, aged 11 years, seemed to be progressing satisfactorily although the abscess was not decreasing in size. On the fifth day he suddenly collapsed and showed signs of general peritoni-

tis. Immediate operation was done, drainage was instituted locally and suprapubically, and the patient recovered. Jopson and Pfeiffer[15] report three patients with perforation of an appendix abscess into the free peritoneal cavity, although none occurred in patients hospitalized under the conservative regime. All had had sudden exacerbations of their abdominal symptoms with evidence of a violent peritonitis. Two had also shown evidence of circulatory collapse at the time of the rupture. Operation was done in one case two days after rupture, in another after twelve hours, and in the third in four or five hours. Only the last patient survived. They emphasize that when an abscess perforates into the free peritoneal cavity immediate operation is imperative.

Continued Spread of the Peritoneal Infection.—The process of inflammation within the abdomen may fail to limit spread of infection from a perforated appendix. However, in only 1 of our 77 cases treated expectantly did the infection fail to localize or subside, and the patient, we now recognize, was improperly selected for delayed operative treatment.

If from the onset of treatment abdominal signs do not recede, pain subside, and the pulse rate fall, one should be hesitant to continue the conservative regime. If the abdominal signs are found to be spreading, the pain increasing, or the pulse rising, immediate operation becomes necessary. Bailey draws particular attention to a rising pulse as an indication that the conservative treatment must be abandoned. One need not be alarmed at a maintained temperature for a few days.

WHEN TO OPERATE

The proper time to operate is as important in the successful conclusion of the conservative treatment as is the selection of cases or the management of the regime.

As already indicated, if a localized inflammatory mass is not subsiding or is increasing in size, if an abscess ruptures into the peritoneal cavity, or if continued spread of the peritoneal infection occurs, immediate operation should be done. Fortunately, except in the case of a small percentage of inflammatory masses which do not subside, it is unusual for appendiceal peritonitis not to resolve spontaneously under a properly managed regime of treatment as outlined.

When then should operation be done in the case where the infection is subsiding satisfactorily? Some delay operation only until the peritonitis shows evidence of localizing; others operate as soon as the acute inflammation has subsided, which is usually after eight to ten days of the treatment; while still others wait two or three months after the attack before removing the appendix in an interval stage. We follow the latter form of treatment. Operation in the presence of recent peritoneal infection is too often complicated by the presence of free pus or by such

a mass of adhesions as to make the procedure extremely difficult and hazardous. On the other hand, we have never failed to express surprise at the relative absence of adhesions and the ease of the operative procedure when the appendix is removed three months after the attack.

RESULTS OF DELAYED OPERATION

Mortality.—It is the experience of all who have used the delayed form of operative treatment in appendiceal peritonitis, as outlined, that the mortality rate is significantly reduced. Our seven-year experience with perforated appendices is divided into periods (Table IV). From 1930 to 1934 delayed operation was not used in the early years and in the latter ones only occasionally. This series numbered 122 patients. In the period 1934 to 1937, which includes 126 cases, delayed operation was used in all patients when indicated. The mortality rate in the latter period, 8.7 per cent, was less than one-half that of the 18 per cent of the first period. This reduction was accomplished mainly in the groups of local peritonitis and abscess where the mortality fell from 10 per cent to 5 per cent and from 14.3 per cent to 4.2 per cent respectively.

Guerry[4] in 1936 reported an operative mortality of 1.4 per cent in 139 cases of diffuse peritonitis treated by delayed operation in contrast to a mortality of 10.6 per cent in 94 similar cases treated by immediate operation. Deaver and Magoun[3] report a mortality of 10.5 per cent for all cases with acute appendicitis treated between 1901 and 1905. In the period 1910 to 1914 this fell to 3.7 per cent and between 1915 and 1919 to 4.2 per cent, a reduction which they attributed to the delayed form of operative treatment used in the latter periods. Coller and Potter[8] report a mortality of 9.4 per cent in 87 cases of spreading peritonitis occurring in patients of all age groups treated by delayed operation. Adams and Bancroft[9] in a series of 110 children, 67 of whom had generalized peritonitis, report a mortality of 4.5 per cent using the conservative regime of treatment. Stanton[16] reports 113 cases with well-marked evidences of peritonitis. In 31 with immediate operation the mortality was 42 per cent, while 82 were treated by delayed operation with a mortality of 8.5 per cent. Bunch and Doughty[17] in a series of 512 cases of perforated appendicitis treated conservatively when indicated report a mortality of 9.5 per cent. In similar studies Kirtley and Daniel[18] in a series of 308 cases report a mortality of 16.2 per cent and Sperling and Myrick[19] a mortality of 16 per cent in 175 cases.

Sworn and Fitzgibbon[12] report 487 cases of perforated appendices with palpable mass. One hundred and twenty were operated upon immediately and 69 after a preliminary delay with a mortality of 7.9 per cent. In 298 conservative treatment was used and no operation done at the time with a mortality of 0.68 per cent. Love[13] reports 341 cases of local peritonitis and abscess treated conservatively with a mortality of

3.5 per cent. Immediate operation was done in 1,044 cases of all forms of appendiceal peritonitis with a mortality of 8.8 per cent. Adams[14] in a similar series reports a mortality of 4.2 per cent in 214 cases treated conservatively and 13.2 per cent in 553 cases treated by immediate operation. Bailey[11] in 242 cases of all forms of acute appendicitis treated by immediate operation had a mortality of 1.6 per cent. Of 73 cases with appendiceal peritonitis treated conservatively, the mortality was 1.3 per cent.

Complications.—In our series of 60 patients in whom complete resolution of the peritoneal infection occurred without operation, complications developed in 3, or 5 per cent of cases (Table V). In the series

TABLE V

COMPLICATIONS AND DURATION OF HOSPITALIZATION IN CASES TREATED CONSERVATIVELY WITHOUT OPERATION

	NUMBER CASES	NONFATAL COMPLICATIONS	FATAL COMPLICATIONS AND CAUSE OF DEATH	AVERAGE HOSPITAL STAY NONFATAL CASES
Local peritonitis	13	Ileus 1	Pneumonia 1*	17.4 days
Abscess	50	Recurrence of signs 2	Pneumonia 1*	15.7 days
			Retroperitoneal abscess 1*	
General peritonitis	1		Peritonitis 1*	
Total	64	3 (5%)	4	16.0 days

*Autopsy performed.

of 145 who survived immediate operation, complications were present in 29, or 20 per cent (Table VII). Sworn and Fitzgibbon[12] report 17.1 per cent complications in their group of appendix abscess cases with operation and 6.7 per cent with conservative treatment.

None of our cases have developed subphrenic abscess or pylephlebitis while under the conservative treatment. Bailey states that subphrenic abscess is almost unknown and that the pylephlebitis does not occur under delayed operative treatment.

Duration of Hospitalization.—That the period of hospitalization is not increased is indicated by the average duration of hospitalization in Tables V, VI, and VII. In patients with appendiceal peritonitis treated by immediate operation, the average hospital stay was 26.1 days. In those whose symptoms subsided completely under conservative treatment, it was 16 days. If to this is added a period of 7 days, which is the average period of hospitalization for interval appendectomy, the total is 23 days. In the group in whom operation was unsuccessfully delayed the total period of hospitalization was 29.7 days.

SUMMARY AND CONCLUSIONS

The delayed form of operative treatment in appendiceal peritonitis is based on sound physiologic and surgical principles. It is indicated in all forms of appendiceal peritonitis regardless of age except in the

TABLE VI

COMPLICATIONS AND DURATION OF HOSPITALIZATION IN CASES IN WHICH CONSERVATIVE TREATMENT WAS ABANDONED UNSUCCESSFULLY DELAYED

	NUMBER CASES	DAYS IN HOSPITAL BEFORE OPERATION	NONFATAL COMPLICATIONS	FATAL COMPLICATIONS AND CAUSE OF DEATH	TOTAL AVERAGE HOSPITAL STAY IN NONFATAL CASES
Local peritonitis	2	14.0	None	None	34.5 days
Abscess	10	8.7	None	Pulmonary embolus 2 Subphrenic abscess with rupture through diaphragm 1	28.8 days
General peritonitis	1	5.0	None	None	26.0 days
Total	13	9.2	None	3	29.7 days

TABLE VII

COMPLICATIONS AND DURATION OF HOSPITALIZATION IN CASES WITH IMMEDIATE OPERATION

	NUMBER CASES	NONFATAL COMPLICATIONS		FATAL COMPLICATIONS AND CAUSE OF DEATH		AVERAGE HOSPITAL STAY, NONFATAL CASES
Local peritonitis	65	Residual abscess	3	Peritonitis	2	23.1 days
		Ileus	2	Pneumonia	1	
		Femoral thrombophlebitis	2	Pulmonary embolus	1	
		Parotitis	1	Subphrenic abscess	1	
		Massive collapse lung	1			
Abscess	75	Ileus	4	Peritonitis	3	26.0 days
		Residual abscess	4	Pulmonary embolus	1	
		Fecal fistula	2	Multiple lung abscess	1	
		Femoral thrombophlebitis	1	Retroperitoneal abscess	1	
		Empyema	1	Coronary occlusion	1	
		Purulent bronchitis	1			
General peritonitis	31	Ileus	4	Peritonitis	8	37.2 days
		Residual abscess	2	Shock	3	
		Perirectal abscess	1	Parotitis	1	
				Anesthesia	1	
				Residual abscess with rupture into peritoneum	1	
Total	171	29 or 20%		26		26.1 days

presence of early perforation with widespread signs of peritonitis. Management of the treatment requires absolute rest of the intestinal tract, maintenance of an adequate fluid, mineral, and caloric intake by routes other than by mouth, and careful observation for evidence of continued spread of a peritonitis or rupture of an abscess, in which case immediate operation becomes necessary.

Of 77 patients with appendiceal peritonitis treated conservatively, 60, or 78 per cent, subsided completely in an average of 16 days and were discharged to return for interval appendectomy in 3 months. Thirteen cases failed to subside and formed abscesses which had to be drained on an average of 9 days after admission.

The entire series of 248 cases of appendiceal peritonitis is divided into two periods. In the first, when delayed operation was used only occasionally, the mortality in 122 patients was 18 per cent. In the second period, with consistent use of delayed operation, except in children with generalized peritonitis, this fell to 8.7 per cent in a group of 126 patients. This reduction was accomplished largely in the groups with local peritonitis and abscess where the mortality rate fell from 10 per cent to 5 per cent and from 14.3 per cent to 4.2 per cent respectively.

Failure of reduction in mortality in the group of general peritonitis cases is caused by the fact that in both periods immediate operation was used in all children with diffuse peritonitis. Application of the principle of delayed operation to children with general peritonitis the same as to adults, even though such treatment is recognized to be less efficient in children, we feel would have reduced the mortality in the diffuse peritonitis group.

The complication incidence in survivors of delayed operation was 5 per cent and of immediate operation 20 per cent. The average period of hospitalization of survivors in the group which subsided completely under the conservative regime was 16 days and in the group with immediate operation 26 days.

The reduction in mortality, incidence of complication, and duration of hospitalization in patients with appendiceal peritonitis treated by delayed operation justifies the more widely accepted use of this form of treatment.

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END RESULTS FOLLOWING THE REMOVAL OF AN "INACTIVE" APPENDIX

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IT IS generally agreed that the treatment of the acutely inflamed appendix is its immediate removal. However, it has happened to every surgeon that occasionally, while the history and physical findings have seemed typical of acute appendicitis, an apparently normal appendix was found at operation; furthermore exploration of the abdomen may have failed to reveal any other diseased organ to account for the physical findings. The pathologist, finding no gross or microscopic evidence of inflammation, usually diagnoses such appendices "inactive."

On trying to rationalize the whole situation after operation, the surgeon may wonder if the patient really had appendicitis and if the operation was necessary. However, careful preoperative and operative examination excluded other possible causes. Even laboratory and x-ray examinations of the patient after appendectomy may fail to detect any pathologic condition to account for the symptoms. As a result, the surgeon usually resigns himself to wait and see if the patient develops any symptoms following operation.

The purpose of this paper is to report the results following appendectomy where the patients had the history and physical findings suggestive of acute appendicitis, yet the gross and microscopic examination of the removed appendix failed to show any inflammation (inactive). In this study appendices removed incidentally during the course of some other operation were not included, nor were cases of acute appendicitis treated conservatively at first and then at a later date the appendix removed after the acute inflammation had subsided.

One hundred and forty-three such cases were available for study. These occurred between Jan. 1, 1931, and July 1, 1937. In this same period a total of 1,770 cases of appendicitis was recorded at the University of Minnesota Hospitals. Fifty-three of the subjects were males and 90 females. One hundred nineteen of the cases (83.3 per cent) were under 20 years of age. There was no increased incidence of the disease during any particular time of the year.

All the patients complained of pain in the right lower quadrant of the abdomen. Eighty-one of the patients were nauseated and 71 gave a history of vomiting. Twenty-four had anorexia. Three patients complained of diarrhea, and another 3 had burning and pain on urination.

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Seventy-eight of the patients were seen during an acute attack, of which the duration in 34 cases was less than forty-eight hours. Seventeen of these 78 cases gave a history of one or more previous attacks.

Sixty-five of the 143 cases complained of recurrent attacks of pain in the right lower quadrant of the abdomen. Because the symptoms were not so acute, more detailed study could be given to the patients in this group. Eighteen patients had x-ray studies of the kidney, ureter, and bladder (KUB); 3 were given barium enemas, 1 had a Graham-Cole dye-excretion test of the gall bladder, and 3 had gastrointestinal studies; 9 had x-rays of the chest and 9 had cystoscopic examinations. The results of all these studies were negative.

Physically, there was pain, tenderness, and rigidity in the right lower quadrant of the abdomen in all cases. In 30 per cent of the cases there was a suggestion of rebound tenderness in that region. On rectal examination, one-fourth of the cases complained of tenderness on the right.

The temperature of these patients ranged from 99.2 to 99.4°. In only 1 was the temperature as high as 101.2°. The number of leucocytes in the blood was under 15,000 in all but 9 cases. The highest leucocyte count was 23,500. The total number of polymorphonuclear leucocytes in the blood smear was usually under 80 per cent. In 14 cases it was over 80 per cent, and in 1 the total count was 94 per cent.

Examination of the urine was negative in all but 8 cases, in which albumin, red or white blood cells were found. Seven of these patients had x-ray studies of the kidneys, ureters, and bladder (KUB), and 5 were examined with the cystoscope with negative results.

An appendectomy through a McBurney incision was performed in all cases. There were no deaths in this series.

RESULTS

A letter was written to each patient asking him to come back to clinic for a checkup or to write in his own words how he had felt since his

TABLE I

		TOTAL PER CENT	PER CENT AVAILABLE FOR CHECKUP
Total number of patients	143	100.0	
Failed to reply	41	28.6	
Available for study	102	71.4	
(a) Examined at clinic	18		
(b) Replied by mail	84		
Recurrence of same or other symptoms	12		11.7
Felt well since operation	90		88.3
Felt well less than 1 yr.	5		2
Felt well 1 to 2 yr.	19		12
Felt well 2 to 3 yr.	20		16
Felt well 3 to 4 yr.	26		19
Felt well 4 to 5 yr.	27		14
Felt well over 5 yr.	46		27
	143		90

operation. It was very desirous to contact patients who had been operated upon four or five years previously. A summary of the results is given in Table I.

The salient points of this follow-up are:

1. Of a total of 143 patients from whom an inactive appendix had been removed, 102 replied by letter or were examined in clinic as to their condition since operation. Of these, 90 were well and 12 had had recurrence of the same or other symptoms.

2. Seventy-three of 143 patients had been operated upon four or more years before. Forty-one of these 73 patients were followed; 32 (78.1 per cent) were well, and 9 (21.9 per cent) were not. Those not well were as follows:

TABLE II

HOSPITAL NO.	SEX	AGE AT APPENDECTOMY	DATE OF APPENDECTOMY	PRESENT COMPLAINT OR DIAGNOSIS
595239	F	11 yr.	Aug. 23, 1930	Still has pain right lower quadrant, especially at pregnancies.
56041	F	17 yr.	Oct. 18, 1930	Still has pain right lower quadrant, especially at pregnancies.
58012	F	21 yr.	March 6, 1931	Pyelonephritis.
634048	M	32 yr.	March 9, 1933	Chronic duodenal ulcer controlled by diet.
632617	F	22 yr.	Nov. 8, 1934	Low back pain; retroverted uterus
616262	M	44 yr.	April 16, 1933	Syphilis
614938	F	17 yr.	June 17, 1933	Bilateral cystic ovaries and right salpingitis (?)
615951	M	29 yr.	April 6, 1933	Psychoneurosis (?)
630245	F	24 yr.	Aug. 29, 1934	Chronic cholecystitis; cholecystectomy, June 24, 1935; still has right lower quadrant pain.

ANALYSIS OF RESULTS

There are several possible sources of error in this type of study:

1. Personal error: There is always a personal error in tabulating results from a chart if the author has not seen the patient.

2. Accuracy of the pathologic diagnosis: At the University of Minnesota Hospitals, two sections are taken through the intact appendix in this type of case; one through the distal third and one through the proximal third. No inflammation was seen in either section of the appendix.

3. The possibility that the acute inflammation in the appendix had subsided: This is remote as half the cases were operated upon within forty-eight hours after the onset of the acute attack.

4. The size of the series.

5. Manner of the follow-up: All the patients should be interviewed and examined personally.

In the available literature no report of a similar study has been found. C. W. Mayo reported satisfactory relief from symptoms after appendectomy in more than 70 per cent of patients with indefinite pain

on the right side of the abdomen where a careful preoperative examination excluded other palpable causes. The pathologic changes in the appendix were graded "chronic 1, 2, or 3" or "chronic catarrhal."

The question always arises in this type of study as to whether the patient's symptoms were due to a disturbance in the appendix. Clinically there seemed to be no question as to the diagnosis. At least three examiners saw each patient and agreed from the history and physical findings that the patient had appendicitis. "Mass psychology" may have influenced the diagnosis here as in other diseases. If the findings were atypical at all, the general opinion was probably in favor of an appendectomy knowing how treacherous an inflamed appendix may be.

It may be questioned how significant the finding is that 78.1 per cent of the patients followed four years or more after operation were well. The majority of patients from whom an inactive appendix was removed may have recovered without surgical treatment. Certainly a large number of patients who suffer a low grade attack of acute appendicitis may recover from the acute attack without an operation. However, it is not known what percentage of such patients remains well four years or more or what percentage suffers recurrent attacks. No one has apparently thought it worthy of investigation to see what percentage of persons who have had an appendectomy for an acutely inflamed appendix has had symptoms after operation. This would be an important control study.

RATIONALIZATIONS CONCERNING THE "INACTIVE" APPENDIX

How to explain the symptoms in these cases where an inactive appendix is removed is a difficult one. Because the patient obtained relief from symptoms after removal of an organ does not necessarily mean that that organ was the prime seat of the disorder unless relief occurred with frequent consistency. However, if one does not believe the appendix to be at fault, one still has to explain the symptoms in these cases. It is always possible, of course, that some occult lesion elsewhere in the body was the cause of the symptoms, such as duodenal ulcer, pyelonephritis, pelvic inflammatory diseases, etc. The fact that this occurred in only 21.9 per cent of the followed cases makes some other explanation necessary unless one assumes that the primary lesion eventually healed in the other 78.1 per cent.

From the work of Wangensteen and co-workers, it is known that obstruction and infection are two important factors in the etiology of acute appendicitis. However, there was no histologic evidence of inflammation in these inactive appendices. Unfortunately, accurate observations of appendicoliths or other obstructing mechanisms in the appendix were not made. Experimental studies of the cecal appendage of the dog show that infection without obstruction and obstruction without infection (at least, obstruction of the washed cecal appendage of the dog) do not cause appendicitis.

While the appendix and cecal apex are comparable structures, the dog does not have a true vermiform appendix. Only man and the higher primates (chimpanzee, gibbon, gorilla, and orangutang) possess a true vermiform appendix. The rabbit has a vermiform-like cecal appendage but an organ which yet does not satisfy the differential characters of comparative anatomists for a true vermiform appendix (unpublished data, Wangensteen and others). Wangensteen, Dennis, and Buirge have studied the behavior of the vermiform appendix or cecal appendage of man and a large number of animals. Only in man, chimpanzee, and the rabbit does the chief function of the appendage appear to be secretory. In all other animals examined, including the monkey, absorption rather than secretion of fluid appears to be the more important function. In the rabbit, obstruction of the appendix causes a rapid fluid secretion. In ten to fourteen hours the intraluminal pressure may have increased because of fluid production to the extent that rupture occurs. Microscopic study of the cecal appendage of the rabbit showing perforation in consequence of obstruction reveals a histologic reaction similar to that occurring in suppurative appendicitis in man.

In man the appendix shows a definite resistance to intraluminal perfusion, which amounts, on an average, to 38 c.c. of sustained water pressure in a normal appendix, and about 73 c.c. in acute appendicitis. The resistance of the appendix to intraluminal perfusion in patients with appendicostomies is about 47.7 cm. of water pressure. It is interesting that long-sustained increases of intraluminal pressures in the vermiform appendix exteriorized by appendicostomy occasionally cause the patient abdominal pain, nausea, or vomiting. Some of these patients even exhibit slight febrile and leucocytic responses (Wangensteen and others). From these experiments it is a logical conclusion that resistance to the outflow of the appendix is very likely the cause of pain in cases of acute appendicitis.

Wangensteen and co-workers have also shown that experimentally obstruction alone will cause acute appendical inflammation in man, the rabbit, and chimpanzee. In man, obstructive concretions are found in the lumen in a large percentage of acutely inflamed appendices. That acute suppuration of the appendix may occur on the basis of obstruction in the absence of such concretions appears likely in that the appendix apparently behaves like a potential closed loop; that is, there is something akin to a sphincter-like mechanism at its junction with the cecum which interferes with evacuation of material which has found its way into the appendix. When the sphincter-like mechanism at the base of the appendix resists evacuation of its contents, the lumen distends, the intraluminal pressure increases, and the chain of events leading to inflammation of the wall begins.

How the appendix may become totally obstructed in other manners than by appendicoliths or foreign bodies in the lumen of the appendix

has yet to be explained. Wangensteen has suggested that swelling of the mucous and submucous lymphoid tissue may bring about obstruction of the lumen, and also that augmentation of normal physiologic obstruction to emptying by reflex nervous cases may be an etiologic factor.

This last factor may be an etiologic explanation of the symptoms in cases where an inactive appendix is removed. The resistance to outflow due to functional disturbances might reasonably explain the pain. Certainly one would expect to find few if any signs of inflammation in such appendices. Since in other organs functional disturbances may give rise to signs and symptoms similar to those associated with organic lesions, why not in the appendix? Since the appendix serves no useful purpose, the best treatment for disorders of that organ, whether functional or organic, is appendectomy.

Every conscientious surgeon deplores the promiscuous removal of the appendix. However, just because the removed appendix fails to show evidence of inflammation does not mean that the operation was unnecessary. The significance of altered function of the appendix is an important consideration for the pathologist as well as the surgeon. Experimentally, the whole problem of the pathogenesis of appendicitis would appear to start with determination of the various manners in which the appendix may become obstructed. It is suggested that the symptoms in some cases where an inactive appendix is removed may be due to a functional disturbance of the appendix. This possibility demands factual proof.

SUMMARY

Of 143 cases in which an inactive appendix was removed, 102 were available for a follow-up study. Of these, 90 were well and 12 suffered from the same or other symptoms. Of 73 patients who had been operated upon four or more years previously, 41 were followed: 32 (78.1 per cent) were well and 9 (21.9 per cent) were not.

Occasionally the appendix, when removed during what appears to be an acute attack of appendicitis, will show no evidence of acute inflammation. In the light of the results of this follow-up study the surgeon need not feel apologetic over the removal of an appendix inactive from the point of view of the pathologist. It seems quite probable that many patients recover from appendicular colic and fail to exhibit microscopic evidence of inflammation of the appendix.

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TUBERCULOUS PERITONITIS

A Speculation on the Cause of Improvement Following Surgery

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WHEN the words tuberculous or tuberculosis are mentioned, the first thought to come to mind is infection of the lungs, the treatment of which is mostly medical except in the late stages. Tuberculous peritonitis, which may or may not be the primary disease, has for the basis of its treatment surgery. A review of this subject is deemed most timely because of a complete study made on a patient recently operated upon at this hospital.

Faulkner and Everett stated that it was not until the advent of modern surgical methods had led to frequent laparotomies that anything like an adequate idea of the frequency of tuberculous peritonitis and of its various clinical and pathologic manifestations was reached. The first adequate description of the disease in the form of a case report was given by Morgagni in 1744. In the same century two other cases were reported in this country, one by Johnson in 1779 and another by Walther in 1785. The first half of the nineteenth century brought descriptions of the pathologic anatomy by workers interested in tuberculosis (Bichot, Laennec, Boyle, and Louis).

In 1872 Spencer Wells performed a laparotomy in a young woman for what he believed to be an ovarian cyst. He found the typical picture of the ascitic type of tuberculous peritonitis. He simply removed the fluid and closed the abdomen; the patient recovered and was well twenty years after the operation. Thus, we see that surgical treatment of tuberculous peritonitis was found because of a mistaken diagnosis. It was König, in 1884, who noted that four patients recovered following laparotomies as the method of treatment. Subsequent studies by König indicated that laparotomies were curative in this disease. In 1887 Van de Warker in this country, advocated laparotomy in tuberculous peritonitis because of "its possible benefit."

Since Sir William Osler published his monograph, there has been increased study of this disease as indicated by the number of articles appearing in the literature since 1890.

ETIOLOGY

The organisms causing tuberculous lesions are the tubercle bacilli, the human and bovine type being the chief offenders in man. In the United States the death rate from tuberculosis decreased from 195 per

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100,000 in 1900 to 72 per cent 100,000 in 1929. Formerly, tuberculosis stood first as the cause of death, but it is now in seventh or eighth place. The most common method of infection is by inhalation, but transmission by the digestive tract is most common for tuberculous peritonitis. Such infection may take place by direct contamination of food or by indirect contamination by the fingers, flies, and the animals producing milk. Zinsser quoted the U. S. Public Health Service as finding 6.72 per cent of market milk infected with the tubercle bacillus. Ashley found that 50 per cent of cases of abdominal tuberculosis are due to the bovine bacillus.

C. H. Mayo wrote, in 1929, that we have gradually learned of the bovine type and its method of transmission to man through milk. Thus, tuberculosis of the bovine type represents at least 25 per cent of the tuberculosis in children and is found on the farms and in small towns and villages. He stated that England and Scotland have paid greater attention to bovine tuberculosis and believes their statistics are better than ours. Tuberculosis of the tonsil, cervical lymph glands, appendix, and mesenteric lymph glands may be foci for peritoneal tuberculosis. Colt and Clark stated that when a local tuberculous process is very active and a caseous gland ruptures, local or general tuberculous peritonitis ensues.

INCIDENCE

Faulkner and Everett found an incidence of 2.8 per cent in 7,000 autopsies. These authors agreed that the disease is more common in females. C. H. Mayo explained that involvement of the pelvis in women may be caused by tuberculosis of the oviduct. He wrote that reports of necropsies showed more than twice as many males as females affected with tuberculous peritonitis, but probably three times as many females as males are operated upon for this condition. Osler believed the disease more common among colored people, but the consensus now, according to literature, is that the frequency is about the same in the two races.

The greatest age incidence occurs between the ages of 20 and 40 years, but it may occur at any time of life. There is an absence of a history of tuberculous contact in these patients, according to Faulkner and Everett.

PATHOLOGIC TYPES

Eliason wrote that from the standpoint of the surgeon the disease is best indicated as being of the exudative or wet type, the adhesive or dry type, and the suppurative type. Further classification into the acute and chronic is necessary as it is generally conceded that the fulminating or acute types are not amenable to surgical intervention.

SYMPTOMS AND DIAGNOSIS

Tuberculosis of the peritoneum may present a variety of clinical manifestations, depending on the acuteness or chronicity of the type. Osler pointed out that symptoms may be latent. Faulkner and Everett showed the chief complaint of pain was abdominal or lower abdominal in 95 of 161 cases. Other symptoms given as the chief complaint were varied, but abdominal swelling and weakness were the most frequent.

The acute form may occur suddenly, especially in children, with chills, temperature of 101 to 105° F., headache, vertigo, malaise, nausea, vomiting, abdominal pain, distention, tenderness, rapid pulse, anorexia, leucopenia, and rapid ascites as the usual symptoms. Rapid ascites is a significant diagnostic point, according to Eliason. The digestive symptoms are far from specific, according to Faulkner and Everett. Nausea and vomiting are inconstant. Constipation or constipation alternating with diarrhea is stressed by some authors. We know, also, that other diseases may cause symptoms of the digestive tract similar to these. Weakness and susceptibility to fatigue should always arouse one's suspicions of tuberculosis. Urinary symptoms, such as dysuria, frequency, and hematuria, should be investigated for a tuberculous origin. Murphy stressed the fact that in the serous ascitic variety of the disease alternate remissions and exacerbations of symptoms are frequently observed.

Faulkner and Everett conclude from their own experience "that the presence of abdominal swelling and distention, particularly if there is definite ascites and a history of pleurisy, associated with abdominal pain or pelvic pain which is worse at the menstrual periods, with a low grade fever, higher toward evening, and loss of weight, with some digestive symptoms especially constipation, and with dysuria or frequency, or both, constitute a syndrome which should strongly arouse the clinician's suspicions of peritoneal tuberculosis."

About 27 per cent of these can be diagnosed preoperatively. Those most easily diagnosed are the ones with ascites and abdominal tenderness. Examination by pelvic route may show palpable masses, tenderness, or distention. The diagnosis is very easily missed because the condition is not kept in mind.

TREATMENT

The proper treatment is the combination of general treatment of tuberculosis and the intelligent use of surgery. General treatment consists of the proper dietary and hygiene, and the employment of ultraviolet rays.

Surgical treatment has to offer several procedures of laparotomy alone, laparotomies with eradication of focus, paracentesis with removal of fluid, paracentesis with removal of fluid followed by the injec-

tion of air or oxygen. Tuberculous peritonitis should never be drained because of the possibility of a fecal fistula's developing.

Stein, in 1934, recommended oxygen pneumoperitoneum whether the condition was primary or of genital or intestinal origin. This method may be combined with the use of iodized oil instillation. Both are of diagnostic and of therapeutic value.

Savage, in 1928, wrote that treatment of tuberculous peritonitis could be accomplished by administration of ether anesthesia. He stated that surgeons frankly admit that results in this disease are out of all proportion to the surgical work done and have advanced one or more of the following theories to explain their cures: trauma, liberation of fluid with or without drainage, decrease in intra-abdominal tension, admission of sunlight, atmospheric air and the actinic sun ray. He remarked that it was strange that no one had considered anesthesia. Savage reported seven patients, one of whom died, all having been treated by closed ether anesthesia for thirty to fifty minutes without operation. The fatal case was puzzling to the author so he looked to a pathologic explanation. He found that there are three stages of the disease: the first stage is one of passive congestion; the second is that of small, isolated, glistening tubercles; and the tertiary stage is that of the large gray tubercule which tends to caseate and coalesce. Investigation by the author showed that the patient with the tertiary stage was the one who died.

Savage saw a girl aged 4 years and 7 months in consultation with Dr. George B. Twitchell, of Cincinnati, Ohio. This girl had been ill from November, 1914, to March, 1915. Twitchell, who did not believe Savage's ideas on the treatment of tuberculous peritonitis, agreed, after they had talked to the girl's mother, to try the ether anesthesia. It was agreed that if improvement did not come at once, surgery would be resorted to. The child was given ether anesthesia for thirty minutes. The verbatim record of the improvement follows: "On the morning following the anesthetic, so the mother stated, she ate more than she had for one week. During the ten days immediately following the anesthetic, indiscretions in diet necessitated free purgations on two occasions. Her periods of sleep lengthened; on the fourteenth (March, 1915), four days following the anesthetic, she slept the entire night and so continued. Her restlessness and night cries ceased and she assumed a natural prone position in bed. Her temperature did not go above normal after the anesthetic. Her convalescence was not interrupted and her recovery was complete."

Savage believes that chloroform would give the same results but that gas anesthesias will to a less extent. He concluded that the earlier the anesthesia is applied, the better are the results, but when there is no response to the ether anesthesia, no hope for relief can be expected from any form of treatment.

The following case reports have been taken from the files of the Robert Packer Hospital.

CASE REPORTS

CASE 1.—A white female, aged 65 years, was admitted to the hospital Nov. 27, 1937, with the chief complaints of loss of weight and distress or heavy feeling in lower abdomen. She stated that since July, 1937, she had been losing weight. Her normal weight was 145 pounds and her weight one month before admission was 135 pounds. Her appetite had been poor through the summer months but had improved some at the time of admission. Her bowels had been regular and she had had no spells of diarrhea. She had a distressed feeling in the lower abdomen which she said was not pain but a feeling of distention or gas; this distress was not associated with or made worse by intake of food and was worse at times and then seemed to improve. She said that she did not have any ambition and felt rather weak at times and that for six weeks prior to admission she had been having severe night sweats.

Her menopause had occurred twenty-eight years before and she had noted no spotting since that time. Since the onset of the present illness, she had had some vaginal discharge which required wearing a napkin irregularly. She had borne one child during thirty-seven years of married life. She had no frequency of urination, but had nocturia once a night regularly. There had been some attacks of dizziness and palpitation.

Examination showed a well-developed and nourished female. The chest, spine, and extremities gave no findings. The heart rate was 87 and the blood pressure was 180 systolic, 90 diastolic. There was a loud systolic murmur but no evidence of decompensation. The abdomen gave an impression of a doughy mass in the lower portion, and pelvic examination gave the impression of the same sort of doughy mass, which was suspected of being malignant.

Laboratory studies revealed: Urine, specific gravity 1.020 to 1.027, acid, and occasional pus cells; red blood cells, 4,420,000; hemoglobin, 84 per cent or 13 gm.; white blood cells, 11,600 with 88 per cent polymorphonuclears and 12 per cent lymphocytes. Her serology was negative, while the blood urea was 55 mg. and the blood sugar 118 mg. per 100 c.c. Widal and undulant fever agglutination tests were negative.

On Nov. 30, 1937, laparotomy was performed. At operation it was found that the pelvic organs and intestines in the lower abdomen were densely adherent by innumerable, rather firm adhesions. The parietal peritoneum and that covering the organs was profusely studded with small discrete pearly tubercles; no excess peritoneal fluid was found. No attempt was made to free the adhesions. The diagnosis from this laparotomy was tuberculous peritonitis. The anesthesia was changed from ethylene to ether and was administered for thirty minutes. The wound was closed in the usual way.

Following the operation, the patient's convalescence was uneventful, with very little distention. On Dec. 10, 1937, a Mantoux test was done and was found to be one plus positive. Again, on Dec. 15, 1937, a bovine tuberculin test was performed. On the day following the administration of the test, the patient was stricken by a severe chill followed by a temperature rise to 102.8° F. The temperature gradually subsided and was down to its former level, ranging between 98 and 99.5° F. in twenty-four hours. During this reaction the patient suffered anorexia, weakness, malaise, and severe sweats. In two or three days all symptoms were gone. The bovine test was read as three plus positive. It was concluded from the character of the rections of these two tuberculin tests that this patient's infection was of the bovine type.

Roentgenograms of the lungs were taken Dec. 18, 1937, to rule out any pulmonary focus. The films showed increased bronchovascular markings but no evidence of parenchymal lesions of the lungs. The aortic arch was shown to be calcified.

This patient was seen and examined in the clinic on Jan. 26, 1938, at which time she had regained much of her weight and strength.

CASE 2.—A white female, aged 13 years, came to the clinic on July 28, 1928, complaining of pain in her abdomen of twelve hours' duration. She was awakened late at night by pains in her lower abdomen. The pain localized in the midline. The patient had no nausea or vomiting, but a progressive distention of the abdomen had been noticed. For four or five days previous to her present illness, she had not been feeling up to par and for one week previously she had had a profuse vaginal discharge.

Her temperature on admission was 101° F. with a pulse rate of 124. The cheeks were flushed and the patient appeared acutely ill. The abdomen was markedly distended and extremely tender. She was considered to have an acute appendicitis which was probably perforated. Operation was advised and a diffuse tuberculous peritonitis was found. The abdomen was closed and closed ether anesthesia was given for thirty minutes. The patient was discharged Sept. 23, 1928, much improved. Five years later (1933), the patient returned with the same set of symptoms. This time she was considered to have a recrudescence of the tuberculous peritonitis although her abdomen was not opened; closed ether anesthesia was administered for one hour and recovery was prompt. The patient has since been in good health with no recurrence of symptoms.

On Dec. 27, 1937, this patient was asked to report to the clinic for a check-up. She stated she had been symptom free and that she had had no recurrence of the symptoms of tuberculous peritonitis. About one year ago she had married and shortly after developed an infection in her left kidney. A left nephrostomy was performed at another hospital. In view of this recent left pyonephrosis, we were anxious to know whether or not it was of tuberculous origin. A communication from the hospital in which she was operated upon indicated that one quart of pus was drained from this kidney, that the pus was sterile by culture, and that kidney function was markedly diminished.

CASE 3.—This white female, aged 31 years, came to the clinic Jan. 19, 1927, with the complaint of bloating of the abdomen, noticeable for about three weeks before admission. She had vomited a few times and her bowels had not moved since the onset of the present illness. She had some pains in both groins. Headaches had been severe and frequent during this present illness. She had a nocturia of three or four times, but no frequency during waking hours. At this admission she stated she had been in this clinic eight years previously, at which time she was operated upon and was told that she had tuberculous peritonitis.

Examination at this second admission showed the abdomen to be quite distended and slightly tympanitic. There was general tenderness, especially so in the right lower quadrant. The abdomen was fluctuant. Vaginal and rectal examinations gave no clue to the diagnosis.

In view of the previous history of tuberculous peritonitis, the patient was considered to have a recrudescence of the tuberculous lesion. She was given closed ether anesthesia for thirty-five minutes and five days later her temperature had returned to normal from an admission temperature of 102.4° F.

She returned for a check-up on March 2, 1927, with her condition much improved and with a weight gain of six pounds. Again, on April 4, 1927, she was seen and stated that she felt excellent; her abdomen was flat and she was allowed

to return to her work. The family physician of this patient reported that she was in good health four or five years ago but that he had been unable to follow her since that.

CASE 4.—A white female, aged 24 years, came to the hospital July 26, 1926, complaining of pain in the right upper abdomen. For years she had had attacks of acute indigestion at infrequent intervals. She claimed she had had an attack of jaundice which lasted one month. She had no pain at this time but considerable gas and indigestion. There was epigastric soreness one month before admission. The day before admission she had a sharp pain in the right upper quadrant, following which she developed soreness in the lower abdomen. The patient complained of nausea but no vomiting. She was severely constipated. Examination at this time showed nothing except slight upper abdominal tenderness. Her temperature on admission was 98° F., but within twelve hours she developed a chill and her temperature rose to 102.4° F. Laboratory tests gave no clue to the diagnosis.

In view of the history and findings a laparotomy was performed under ethylene anesthesia on July 31, 1926. Through a right rectus incision the abdomen was explored and found to be filled with straw-colored free fluid. All peritoneal surfaces were studded with myriads of small tubercles and a large mass was located in the central part of the abdomen. The abdomen was closed without drainage and without further exploration.

Convalescence was rapid and by the sixth postoperative day the temperature had reached normal levels; she was discharged on the seventeenth day, much improved. A report through her family physician in 1929 indicated that she was in good health and working every day. Because of the death of this girl's family physician, we have been unable to bring her follow-up study to date, as she has moved to California.

CASE 5.—This patient, a white female, 48 years old, came to the clinic Jan. 31, 1927, with the complaints of anorexia and insomnia. She had been well until Dec. 25, 1926, when she suddenly lost her appetite and had some nausea. She had some dyspepsia and was disturbed by a considerable amount of gas. Epigastric discomfort and a heavy feeling in the abdomen were noted. About twenty-one years previous to this time, she stated, she had a similar attack which lasted five weeks. The patient had been eating at boarding houses and hotels. Her menses were regular until six months before this illness, when they stopped. Ten days before her admission there had been a vaginal discharge which was slightly bloody. She had noted a rise in her temperature varying from 99 to 101° F. Insomnia was a prominent complaint.

Examination on admission gave signs enough to suspect a lesion of the right apex. Roentgenograms examined stereoscopically showed the lungs to be clear. Laboratory studies did not aid in making a diagnosis. The patient was suspected of having tuberculous peritonitis and closed ether anesthesia was administered for thirty minutes. She was not subjected to surgery. Twelve days following the anesthesia the patient was much improved and she was discharged from the hospital three weeks after the ether was given.

A check-up examination at the clinic one month after she left the hospital showed much improvement with a normal temperature, increase in weight, and improved appetite. On Nov. 17, 1927, the patient had continued to feel well and had regained her strength.

This patient returned to the clinic Feb. 23, 1938, and she was entirely symptom free. An undulant fever agglutination was negative as was her physical examination.

COMMENT

Since the results of surgery in tuberculous peritonitis are out of proportion to the amount of surgical work performed, there must be some explanation of why these patients improve. Also, it seems likely in some cases that equivalent improvement may be accomplished by the use of closed ether anesthesia alone. A temporary anoxemia produced by the anesthesia might be an explanation for the apparent cures.

The tubercle bacilli are known to be aerobic organisms. Zinsser and Bayne-Jones wrote that the best growth is obtained under conditions allowing free access of oxygen of the air. It has been shown that wax plugs in culture tubes inhibit or reduce the bacterial growth by restricting the oxygen supply. The admission of air by laparotomy, if the peritoneal cavity can be considered as a live culture tube, should enhance the growth of the infecting bacilli. Such a circumstance could hardly be offered as an explanation of improvement after surgery on that basis alone. The application of closed ether, chloroform, or nitrous oxide anesthesia, on the other hand, would produce a temporary anoxemia. Reasoning, again, from the known facts of culturing the tubercle bacilli, the reduced oxygen intake of the body would explain, at least partially, the improvement that follows when ether anesthesia alone is administered.

The chemical study of the tubercle bacillus shows that such compounds as lipoids, fatty acids, acetone-soluble fat, and wax can be isolated. Since the blood carries a large volume of ether vapor during anesthesia, it is possible that some chemical reaction may take place with one or more of these substances to cause permanent injury to the organisms or to produce an oxygen impermeable membrane about them. Such speculations on a chemical basis would tend to uphold further the proposed theory of anoxemia as an explanation of convalescence of tuberculous peritonitis cases following closed ether anesthesia alone or accompanying a surgical procedure.

A study of cases of tuberculous peritonitis which have been operated upon under spinal anesthesia would do much to settle the question of whether inhalation anesthesia is a factor in the improvement.

SUMMARY

The available literature on tuberculous peritonitis has been reviewed.

Tuberculous peritonitis treatment is not medical nor surgical but is a combination of both.

The only article available on the treatment of tuberculous peritonitis by closed ether anesthesia has been reviewed.

Comment has been made as to the possible explanation of the therapeutic action of ether anesthesia in tuberculous peritonitis.

From experiences in this hospital, as indicated by case reports and by the writings of Savage, of Cincinnati, it appears ether anesthesia is a valuable adjunct in the treatment of tuberculous peritonitis.

The postoperative condition of patients who have been operated upon under spinal anesthesia and gas anesthesias, which do not produce anoxemia, should be studied.

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CHRONIC SUBDURAL HEMATOMA

Diagnosis and Treatment

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THE condition known today as chronic subdural hematoma was first described very accurately by Virchow[1] in 1857 under the name of "hematoma durae matris" or "pachymeningitis hemorrhagica interna." He based his studies in most part on autopsy material and concluded the lesion to be spontaneous in origin due to an inflammatory condition which was progressive in type. He described the formation of a very vascular subdural membrane followed by ecchymosis and subdural hemorrhage. He recognized a traumatic type, chiefly in the newborn, but made no comparative studies between the two types. Much interest was aroused by Virchow's work and the condition was studied extensively by Huegenin,[2] Wiglesworth,[3] Robertson,[4] Schuberg,[5] and many others. With few exceptions their chief object was to determine the underlying pathology and little attention was directed to the proper treatment of the lesion. Although Schuberg, as early as 1859, recognized trauma as a cause of chronic subdural hematoma, little attention was paid to its significance until 1914, when Trotter's paper[6] was published. He stated: "It is nearly certain that the veins passing from the brain to the tributaries of the superior longitudinal sinus are always the source of the blood." He explained in detail the mechanism by which this bleeding was produced, and by so doing I believe he gave us a proper conception of the true cause, which is in most cases traumatic.

Putnam and Cushing[7] in 1925 described two classes of subdural hematoma. The first they called a vascular or spontaneous type, and the second, a reactive or traumatic type. The slight histologic difference between the two types is hardly sufficient to justify the distinction.

Spiller and McCarthy[8] in 1899, working on cats and dogs, produced the lesion experimentally by nicking the superior longitudinal sinus sufficiently to produce a subdural hemorrhage. They studied the formation of the hematoma in sacrificed animals at intervals varying from sixteen hours to five weeks and found the new membrane was present in a few days and distinctly formed in five weeks.

Interest in the early diagnosis and treatment of this particular lesion has been greatly stimulated in the past ten years by reports of cases and groups of cases cured by surgical methods. Excellent articles by Jelsma,[9] Kaplan,[10] Gardner,[11] Fleming and Jones,[12] McKenzie,[13] Kee-

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gan,[14] Coleman,[15] Frazier,[16] Furlow,[17] and many others have proved to us that chronic subdural hematoma is a curable surgical condition.

The gross appearance of the lesion as well as the pathology of the sac and its contents has been exhaustively considered in the literature, and I shall not attempt to describe them in detail here. Briefly, the lesion is an encysted clot under the dura and when exposed at operation can hardly be mistaken for any other condition, so characteristic is its appearance. The subdural sac with its liquid and clot contents presents a dark greenish blue appearance through the dura. In the late stages this outer wall may present a greenish yellow appearance due to presence of deposits of bile pigment. In most cases the sac is very extensive,

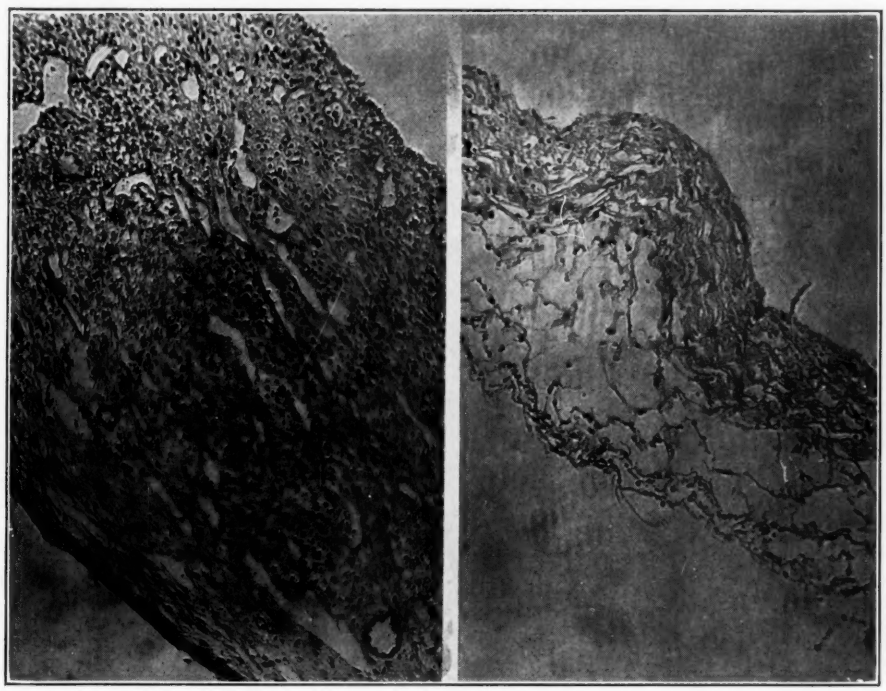

Fig. 1.—(Case 2.) Photomicrographs showing (left) outer wall of sac and (right) inner wall of sac for a subdural hematoma.

covering a large portion of one hemisphere from the frontal pole to the occipital lobe and from the midline down to the Sylvian fissure or below it. The dura usually can be separated readily from the outer wall of the sac. When the sac is opened, dark reddish brown liquid escapes and one may find rusty looking clots in various stages of liquefaction. The inner wall of the sac lies directly over the arachnoid. It is thin and transparent and not adherent to the arachnoid except at its border, especially in the region of the longitudinal sinus. (Fig. 1.) Bleeding may be encountered in this area if an attempt is made to remove this portion of the sac. When the sac and its contents have been removed, the brain presents a flattened appearance and is pushed away from the

dura one inch or more, depending on the size of the clot. The hemisphere appears compressed and does not expand readily to its normal contour. If the hemisphere appears to expand and fill up the space when the sac is removed, a similar lesion on the opposite side should be suspected. (Fig. 2.)

The clinical course of this condition is not always typical, but in a majority of cases the history will disclose the following sequence of events. A history of a trivial blow on the forehead or occiput or a glancing blow over the vertex can often be obtained. This usually is followed by symptoms of slight concussion lasting only for a short period. The patient recovers promptly from the immediate effects and

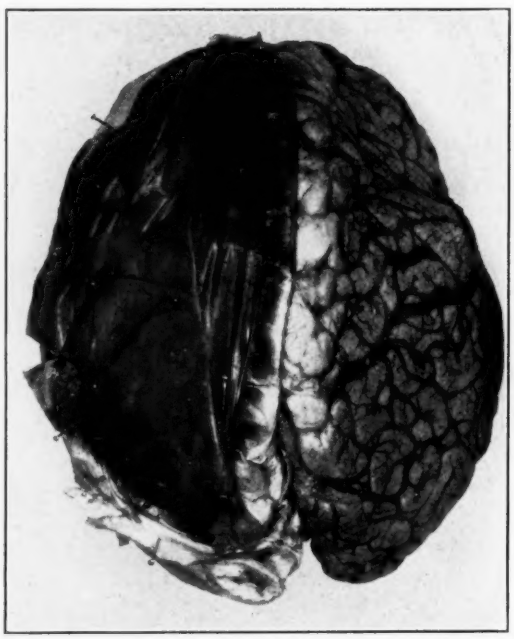

Fig. 2.—(Case of unsuspected subdural hematoma found at autopsy.) Photograph of convexity of brain showing marked compression of the right hemisphere. The dura over the right hemisphere is folded back over the left hemisphere to show the sac of the subdural hematoma.

may resume his former activities with no apparent symptoms. Several weeks or more may elapse before the lesion reaches sufficient size to cause the patient to complain. The first symptoms to appear are those of slowly increasing intracranial pressure of one or both hemispheres. Headache is the first symptom to appear and is usually persistent, although it may vary in intensity from day to day. Generally it increases and is of a throbbing type. Nausea and vomiting may occur along with the headaches.

Along with these complaints, mental changes appear which are often noted by the patient's relatives and friends. The patient becomes dull, inattentive, and forgetful. Personality changes are often noted. Re-

mission and exacerbations are noted from time to time. Later a state of stupor appears, followed by coma. Even in this stage remissions and exacerbations are striking. The patient may pass alternately from coma to consciousness and may appear and act quite normal when conscious.

The following case (Case 1) presents a typical history, as well as the confusing neurological signs often noted:

CASE 1.—E. B., white male, aged 29 years, was admitted to the University Hospital on Aug. 4, 1936, and discharged on Sept. 29, 1936. This patient was in an automobile accident two months before admission to the hospital. He sustained a head injury at the time and was unconscious for a short time. He was treated at home and remained in bed for one week following the accident, at which time he was able to go back to work. He worked for about a month before any symptoms appeared. At this time he developed headache and nausea with blurring of vision and diplopia. His family also stated that he became very forgetful and was at times completely disoriented.

Physical examination on admission showed the patient to be a well developed, slightly obese young man, somewhat disoriented and complaining of severe headache. The positive neurological findings were dull mentality, bilateral anosmia, difficulty with taste for several weeks, bilateral reduction in visual acuity, bilateral choked disks with hemorrhages, slight ptosis of right internal rectus muscle, partial third nerve palsy, diplopia on looking to left, tongue protruding slightly to the left, questionable reduction of motor power in right arm and right leg, slight ataxia in both lower extremities, knee and ankle jerks absent bilaterally, and Oppenheim positive on the left. A diagnosis of subdural hematoma over the right hemisphere was suspected.

On Aug. 27 a small trephine opening was made in the right occipital region over the posterior horn of the lateral ventricle preparatory for a ventriculogram. When the dura was opened in this region, a subdural hematoma was encountered. A right osteoplastic flap was immediately turned down and a large subdural hematoma exposed. The entire sac and its contents were removed and the patient showed marked improvement for ten days, and then began to have headache and blurring vision, and became very drowsy. A spinal puncture at this time showed a clear fluid with increased spinal fluid pressure. Since the patient had improved so promptly following the removal of the sac and had later developed symptoms of increased intracranial pressure, hemorrhage at the site of the operation or a similar lesion on the opposite side was suspected. Since the spinal fluid was clear and the entire sac had been removed on the right side, it was deemed wise to explore the left hemisphere even though there were no localizing signs present.

On Sept. 16 under local anesthesia a trephine opening was made in the left temporal region and a subdural hematoma found over this hemisphere. A second trephine opening was made in the frontal region and the dura and sac opened. The contents of the sac were evacuated by irrigating with salt solution. Both incisions were closed without drainage. The patient made a very prompt, uneventful recovery and was discharged well on Sept. 29, 1936.

The neurological picture is one of generalized increased intracranial pressure. Rarely are there definite focal symptoms. The slow compression of a large part of a hemisphere may and does occasionally produce a gradually increasing paresis to the face, arm, and leg, but rarely is there a paralysis. Jacksonian attacks are practically never seen, but

convulsive seizures may occur late in the course of the illness. Slow pulse is common; cerebrospinal fluid is often under increased pressure and may be either clear or xanthochromic. Early in the course of the lesion the eye grounds appear normal; later, however, there is a mild venous congestion of the disks, followed by a gradual obscuration of the margins and a total absence of the cups.

The symptoms in general conform fairly well to those produced by a slow, progressive, expanding lesion over a large portion of one or both hemispheres without local cortical irritation.

Evidence to support the slow, progressive type of lesion is produced in a case of Coleman,[15] in which an exploration through a small burr opening was negative. There was temporary improvement followed by return of symptoms. An exploration through the same opening eleven days later revealed the lesion which he explained "had evidently moved forward under the exploratory opening in the eleven-day interval."

McKenzie[13] suggested that the increase in the size of the sac was due to a process of osmosis, the sac acting as a semipermeable membrane. As the encysted clot disintegrates, the protein content is changed, thereby increasing the osmotic pressure within the sac. Cerebrospinal fluid then dialyzes through the inner sac wall from the subarachnoid space to restore the osmotic balance. Zollinger and Gross[18] recently have supported this view by some convincing experimental evidence on intact membranes removed surgically. Recurrent bleeding into the sac either from the vein originally injured or from granulation tissue of the dural wall, as suggested earlier by Trotter,[6] Putnam and Cushing,[7] Keegan,[14] and others, probably plays some rôle in producing the slowly expanding lesion.

If a subdural clot is suspected, the diagnosis should be at once verified or ruled out by burr openings over both hemispheres, for in a certain percentage of cases the lesion is bilateral. Air injection is seldom necessary, but it may be helpful when some other lesion is suspected (Fig. 3).

Several surgical procedures have been advocated in the treatment of chronic subdural hematoma: (1) the use of a large osteoplastic flap with the removal of the entire sac and its contents, (2) the evacuation of the clot through small burr openings, and (3) the removal of a clot through a subtemporal decompression opening. Very satisfactory results have been reported in a large number of cases by simple drainage through small burr openings. It is not necessary to remove the sac to effect a cure. Bilateral perforations should be accepted as the method of choice for several reasons: (1) because the procedure is a simple one and can be carried out in a short period of time under local anesthesia, without loss of blood and without shock to the patient; (2) because the one procedure serves to establish the diagnosis, to determine whether the lesion is bilateral, and to evacuate the clot; and (3) because it enables one to evacuate a bilateral hematoma at one sitting. When there

is a well-organized clot present, it may be necessary to reflect a flap to remove it effectively.

Drainage, I believe, is not necessary when the clot has been removed by irrigation and suction through burr openings, unless there is indication at the time of operation of fresh bleeding. In such cases a small tubular drain should be left in each perforation as a precautionary measure, in case of subsequent bleeding.

According to most investigators, cerebral edema is the most important complication to be feared. Postoperative edema was not suspected or proved in any of the fourteen cases reviewed for this discussion.

Postoperative extradural hemorrhage is more likely to occur than cerebral edema, especially when there is no increased intracranial pres-

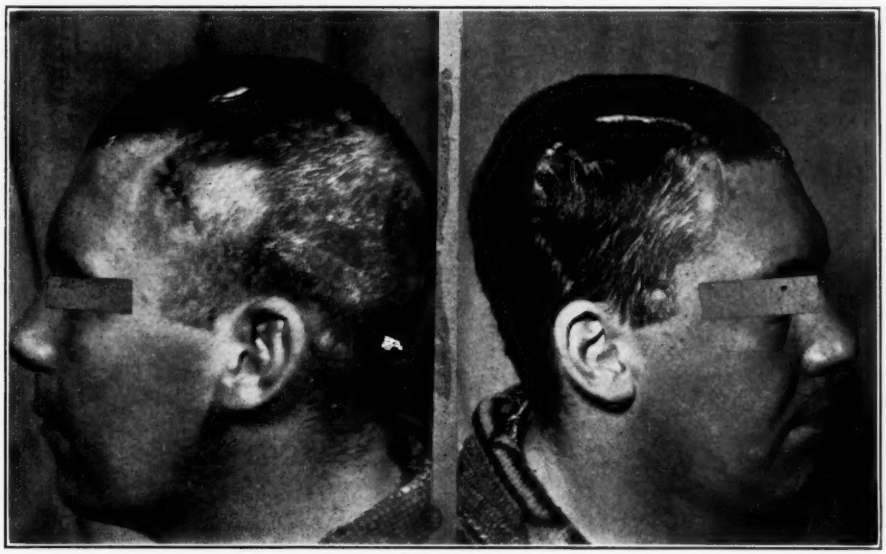

Fig. 3.—(Case 1.) Photographs of patient showing results of operations for bilateral subdural hematoma three weeks following bone flap and one week after trephine operation on opposite side.

sure and when a dead space persists after the evacuation of a clot. This complication occurred in only one of the fourteen cases of our series.

Probably the most important postoperative pathology which exists is the failure of the compressed brain to expand promptly. This, together with the accumulation of fluid in the dead space, has been described by Coleman[15] to be the cause of the stormy, delayed convalescence seen in many cases. This condition has been encountered repeatedly in this series of cases. The duration of the lesion, no doubt, is a determining factor, for in other cases with little compression the convalescence was surprisingly rapid and free from complications. (Fig. 4.)

Marked circulatory changes were noted frequently on the surface of the compressed hemisphere after removal of the sac. The vessels were flattened and did not fill promptly when the pressure was removed. In

one case (Case 2) petechial hemorrhages were found in the brain stem. These circulatory changes due to long-continued, slowly increasing intracranial pressure are perhaps responsible for the long-delayed, stormy convalescence seen in cases which come to operation late in the course of the lesion. (Fig. 5.)

In the following case (Case 2) secondary vascular changes were noted and probably had some bearing on the outcome.

CASE 2.—J. S., aged 52 years, white male, was admitted to the University Hospital on Feb. 13, 1936, and died on Feb. 15, 1936.

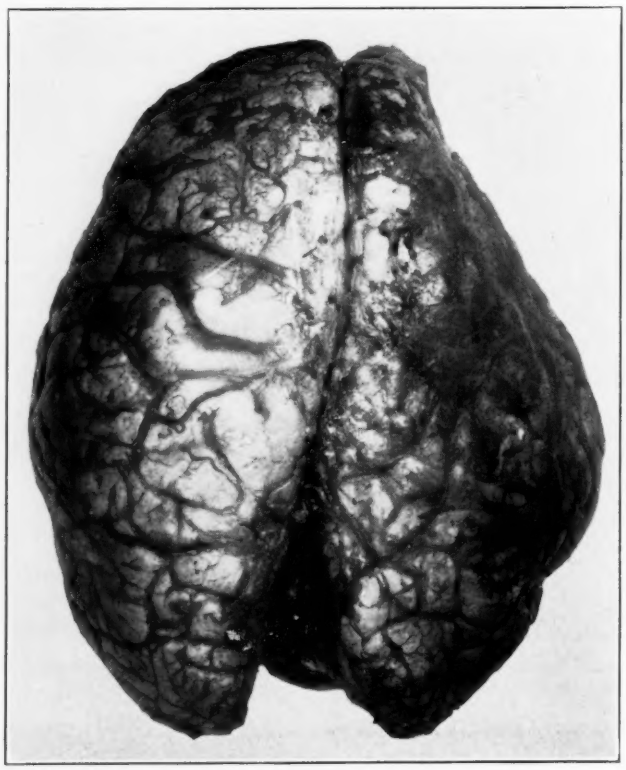

Fig. 4.—(Case 2.) Photograph of brain showing extreme compression of the right hemisphere and secondary vascular changes.

This patient gave a history of having been in an automobile accident two and one-half months before admission. He sustained a laceration over his right eye and was unconscious for about ten minutes. He was taken to a hospital in the vicinity where the laceration was treated and he was discharged. The patient returned to the hospital for dressings on several occasions and apparently was in good health and had no symptoms. He continued his work on the farm until about a week before admission to the hospital. During this period he had only slight attacks of headache and apparently only during the last two weeks had any other symptoms. His family noticed that his memory was failing and that he became untidy and appeared mentally confused at times. One week before admission he became very drowsy and it was difficult to arouse him for several hours. The following day he appeared normal and went about his work. Four days before admission he fell in the yard un-

conscious. He remained in an unconscious state for twelve hours, gradually regaining consciousness until he was able to take fluids by mouth and respond to questions. He was taken to the hospital at this time and later transferred to the University Hospital in Baltimore for treatment. On admission to the hospital he was in a comatose condition and could not be aroused. There were marked signs of dehydration.

On examination the patient's attention could not be kept. He went to sleep very easily, had no idea as to his whereabouts, and knew no one. He had no phobias or hallucinations. Examination of the cranial nerves was as follows: 1, apparently had no anosmia; 2, visual acuity appeared to be normal, but could not be tested adequately; visual fields normal on rough test; slight choking; 3, 4, and 6, pupils round, regular, and central; reacted to light and accommodation; no apparent nystagmus; extra-ocular movements normal; 5, muscles of jaw appeared normal; sensitive to pain and temperature; 7, face was drawn slightly to right, left eyelid seemed to be ptosed; 8, hearing apparently normal; 9 and 10, could swallow; voice normal; pulse slow; 11, negative; 12, tongue protrudes in midline without tremor.

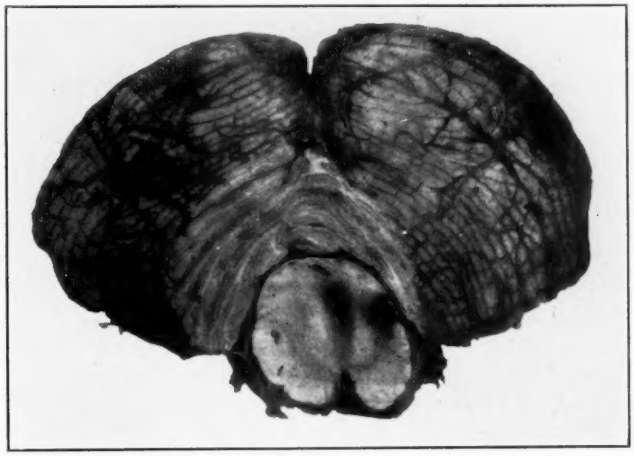

Fig. 5.—(Case 2.) Photograph showing hemorrhage in the brain stem.

The entire left upper extremity was weaker than the right, but there was no tremor, convulsions, or atrophy. The left lower extremity was also weaker than the right.

The physical examination of this patient was as follows: Head: Scalp showed several ridges over parietal and occipital regions on the left. Ears: Showed no tophi, discharge, or mastoid tenderness. Eyes: See under cranial nerves. Nose: Breathing spaces adequate, septum in midline and intact; mucous membranes reddened, slight serous discharge. Mouth: Sordes over lips; tongue coated; teeth carious; throat injected. Neck: Enlargement of lymph glands in anterior and posterior angles. Chest: Expansion limited; percussion note impaired over entire left area, but very slight. Breath sounds vesicular and roughened throughout lung area. Fine râles heard throughout both lung fields. Heart: Appears normal in size and contour to percussion. No shocks or thrills; rate is slow, regular in rhythm and intensity. No murmurs. Blood pressure, 125/90. Abdomen: Flat, soft. No rigidity or tenderness. Liver, spleen, and kidneys not palpable. No masses.

The roentgen examination of the skull showed an average-sized cranium, bones moderately thick. The vessel markings appeared normal. The sella was small,

TABLE OF REFLEXES (CASE 2)

REFLEX	RIGHT	LEFT
Jaw jerk	0	0
Biceps jerk	+	+
Supinator jerk	+	+
Triceps jerk	+	-
Abdominal—		
Upper	+	+
Lower	+	+
Cremasteric	+	+
Knee jerk	+	+
Ankle jerk	+	+
Ankle clonus	0	0
Plantar	Extensor	Flexor
Brudzinski	0	+
No cervical rigidity or Kernig sign elicited		

closed-in variety. There was no radiable evidence of any pathologic change. There was no evidence of fracture.

The laboratory findings were as follows: Urinalysis: Specific gravity 1.030; reaction acid; no sediment, albumin, sugar, acetone; microscopically negative. Blood count: Hb., 99 per cent; Sahli, 15.84 gm.; R.B.C., 5,870,000; W.B.C., 10,450; color index, 0.8; Differential count: Filamentous polymorphonuclears, 61; nonfilamentous, 17; lymphocytes, 14; large mononuclears and transitionals, 8.

A diagnosis of a right subdural hematoma was made. On Feb. 14 a right osteoplastic bone flap was turned down. A large subdural hematoma was found and the sac and its contents removed without difficulty. There was no bleeding at the time of operation, but the whole hemisphere showed a very marked compression and failed to expand when the clot was removed. The flap was replaced with the patient apparently in fair condition. He failed to react, however, following operation. His temperature became elevated and his pulse remained quick. A spinal puncture was done several hours following the operation because postoperative hemorrhage was suspected. The spinal fluid was found to be clear and the pressure was 100 mm. of water. The patient died twenty-four hours after operation.

At autopsy the right hemisphere of the brain showed marked compression and distortion, due chiefly to the inability of the brain to expand after the removal of the subdural hematoma. The parietal and frontal lobes were chiefly affected, but the temporal lobe was also slightly involved. There was a localized area of subarachnoid hemorrhage which extended from the superior temporal convolution to the longitudinal fissure, and anteriorly just in front of the Sylvian fissure and posteriorly to the paracentral lobule. The operative specimen which was removed from the right hemisphere measured 10 by 4.5 by 3 cm. This consisted of a sac of fibrous tissue and had much the same appearance as the dura, except that it was not quite so tough. The membrane was semitransparent and smooth on both surfaces. The mesial wall of the cyst was very much thinner than the lateral wall. The mesial wall had the appearance almost of cellophane; whereas, the external wall was hemorrhagic in color and much thicker.

Section through the optic chiasm showed the right lateral ventricle to be compressed and the septum pellucidum was displaced to the left. The right hemisphere was compressed, as previously described. Both hemispheres showed convolutional flattening due to a marked degree of intracranial pressure. A section through the peduncles showed a vertical hemorrhage 5 mm. below the aqueduct in the midline. This hemorrhage measured 6 by 2 mm. At the periphery of the left peduncle there was another hemorrhage which measured 5 mm. A section through the pons and

cerebellum at the fifth nerve exit showed a small hemorrhage 5 by 2 mm. in the midportion of the pons. Section through the pons at the eighth nerve exit showed a small hemorrhage measuring 3 by 1 mm. on the left side in the lateral portion.

Microscopic examination of the limiting membrane of the subdural hematoma showed a loose, eosin-tinted matrix, embedded in which were numerous round cells, the whole evidently being granulation tissue. There were numerous thin-walled spaces lined by a single layer of cells, but a few well-developed blood vessels were also present. There was condensation of connective tissue fibers on one surface, forming the limiting membrane. Free red blood cells were found in the tissue in small areas. In the microscopic section through the peduncles and aqueduct, the aqueduct contained a few red blood cells. In the middle of the peduncular structure there was an old hemorrhage with some destruction of nerve tissue. The leptomeninges in the interpeduncular space contained numerous pigment granules embedded in the fixed tissue cells of the leptomeninges. There was no cell infiltration. Some recently extravasated blood was also present here. Some perivascular edema was also present in the section.

In the microscopic section through the right superior frontal convolution there was old hemorrhage in the leptomeninges, with hyalinosis of the vessels. The usual thickening and the presence of hemocyteron granules were also noted. The underlying cortex showed no change except edema. There was some lack of clearness in the staining of the nerve cells. The subcortical fiber tracts showed no edema. In the right middle frontal convolution there was a slight amount of old hemorrhage together with edema in the leptomeninges, the presence of a few plasma cells containing pigment, and a few round cells also pigmented in the depths of the sulci. On the surface of the gyri the old hemorrhage was dense, the blood cells disorganized, and numerous small round cells and some polymorphonuclear leucocytes were seen. The amount of free blood pigment gave a dusty appearance. At no place did the hemorrhage break through into the cortex. The limiting membrane of the cortex was a little thickened and did not show rupture. Edema was visible throughout the slide, but there were no areas of softening in the nerve tissue.

Microscopic section of the left middle frontal convolution showed the leptomeninges to be quite edematous. All the blood vessels were engorged but showed no changes in their walls. The underlying nerve tissue was also edematous in both the cortical and subcortical fiber tracts. The basilar artery showed no pathologic change. In the left basal ganglia no change was noted except for perivascular edema.

In this series of 14 cases of subdural hematoma on which this paper is based, there were 2 deaths. One patient (Case 2) died after a flap operation; the other death (Case 10) occurred following the evacuation of the clot through a subtemporal decompression opening. History of trauma was obtained in 12 of the 14 cases. There were 10 males and 4 females in the group. The clot was on the right side in 8 cases and on the left in 6. Only 1 had bilateral clots. The Wassermann reaction was negative in every case.

Choked disks were present in 5 cases. The spinal fluid pressure in the 9 cases without choked disks was increased and in 4 of these 9 cases the fluid was xanthochromic. Headache was the most common complaint and was present in all cases. Mental disturbance was a prominent symptom in 10 cases. Vomiting was not frequently present and occurred in only 4 cases. Paresis of the cranial nerves and extremities was not a common finding. Coma was present in only 2 cases.

A flap operation was used in 7 cases. In 4 the clot was removed through one or more burr openings. Subtemporal decompression openings were used in 3 cases. A secondary flap operation was necessary to remove a solid clot in 1 of the latter group. In the 1 bilateral case a flap operation was done on one side and the clot evacuated through burr openings on the other.

The diagnosis in 10 cases was suspected from the history and verified by burr openings. Air injection led to the diagnosis and localization in 4 cases in which other lesions were suspected. Pineal gland shift was noted in 1 case. (Fig. 6.)

CASE 3.—C. A., white male, aged 24 years, was admitted to the University Hospital on Aug. 1, 1935, and discharged on Aug. 24, 1935.

The patient gave a history of having been in an automobile accident on April 25, 1935, three months before admission. He received a sharp blow on the head just

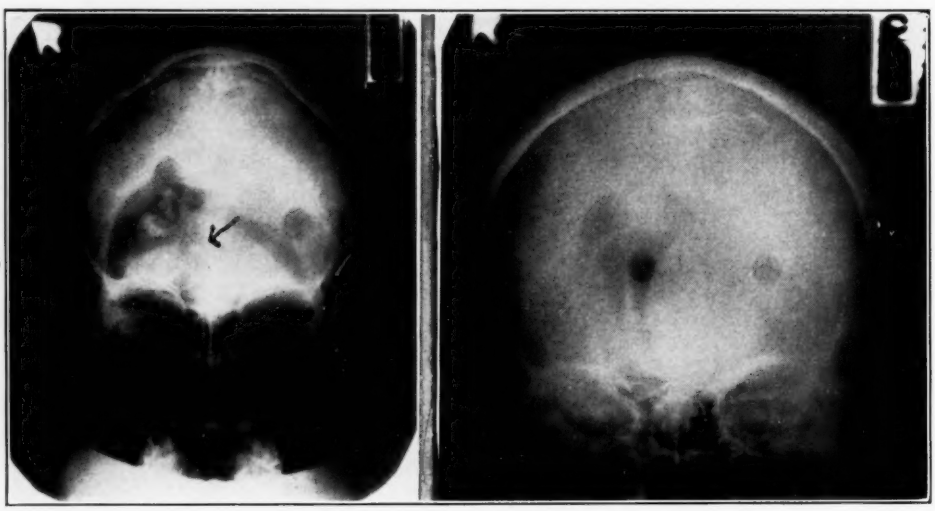

Fig. 6.—(Case 3.) Air injection showing shifting of the ventricular system, distortion of the ventricles, and pineal gland shift.

over the left eye. He was unconscious for about one-half hour and remained in the hospital for three days. After discharge from the hospital, he was able to go back to work and stated that he felt entirely well until July 26, about a week before he entered the hospital. He then developed severe frontal headaches and blurring of vision. On admission, his only complaint was severe headache. The neurological examination was entirely negative except for bilateral choked disks. The tentative diagnosis of subdural hematoma or brain tumor was made.

Three days after admission a ventriculogram was made and showed a marked displacement of the lateral ventricles to the right beyond the midline with compression of the left lateral ventricle. On Aug. 5, a left osteoplastic bone flap was turned down and a large subdural hematoma extending from the frontal pole to the occipital region was found. The sac with its contents stripped readily from the dura and underlying cortex, except a small portion close to the midline in the frontal region where it was apparently attached to the longitudinal sinus. In removing the sac in this area there was considerable bleeding from the tributaries going into

the longitudinal sinus. Before replacing the flap, this bleeding was completely controlled. The entire hemisphere was compressed and showed little evidence of expanding after the sac was removed. The patient made an uneventful recovery and was discharged cured twenty-three days after admission.

CASE 4.—A. E. W., white male, aged 26 years, was admitted to the Maryland General Hospital on Oct. 14, 1936, and was discharged on Nov. 5, 1936.

The patient gave a history of injury to the head in an automobile accident six weeks prior to this admission to the hospital. He was not unconscious, but received first aid treatment at the hospital and was then discharged. He had no symptoms until four weeks after injury when he complained of a generalized headache. Several days later the headaches were more severe and were accompanied by nausea and vomiting. On admission to the hospital the patient was stuporous, but he could be aroused. He was well oriented as to time and place. There was a small scar on the vertex of the head which was well healed. There was a very definite right facial weakness and marked choking of both disks. The neurological examination was otherwise negative.

The spinal fluid was clear with a pressure of 35 mm. of Hg, and a cell count of 3. From the history a subdural hematoma was suspected over the left hemisphere. Under local anesthesia a small trephine opening was made in the upper temporal region on the left side. The dura and cortex appeared normal. The same procedure was carried out on the right side with the same findings. A third trephine opening was made in the occipital region over the posterior horn of the right lateral ventricle, preparatory for a ventriculogram. When the dura was exposed in this area, it appeared bluish in color and when opened there was an escape of dark brownish red fluid. A large amount was evacuated through this small opening and a diagnosis of subdural hematoma was made. No attempt was made to empty the ventricle or to inject air. The sac was allowed to empty itself without irrigation and the incisions were closed without drainage. It was felt at the time that a secondary operation would probably be necessary if there were solid clot present. The patient, however, made a very prompt, uneventful recovery and was discharged in good condition on Nov. 6, 1936.

CASE 5.—M. McK., colored woman, aged 32 years, was admitted to the Baltimore City Hospitals on March 2, 1937, and was discharged on April 21, 1937.

The patient gave a history of a head injury about three months previous to admission. She was not unconscious at the time, but was taken to a hospital where a laceration of the scalp was sutured. She returned to her home and continued her work for almost two months, during which time she had intermittent headaches but not enough to interfere with her work. One month following the injury to her head she was taken to the Sydenham Hospital in a semicomatose condition. Examination showed positive blood and spinal fluid Wassermanns, and a diagnosis of central nervous system syphilis was made. She was transferred to the Baltimore City Hospitals for treatment.

Her neurological symptoms varied from time to time. At one time there was a very definite left facial weakness, mental reaction was very slow, and she complained of headache. On another examination there was a definite right facial weakness, rigidity of the right arm was present, and there were tremors of the right upper and lower extremities. The eye grounds were normal and there were no signs of increased intracranial pressure.

On March 18 a spinal puncture was done. The fluid was clear, with a pressure of 350 mm. of water. One hundred cubic centimeters of fluid were removed and replaced with air through the spinal needle. Encephalography showed marked distortion of the ventricular system. Both lateral ventricles were

displaced to the left of the midline, the left being slightly dilated and the right showing marked compression. The tentative diagnosis of subdural hematoma was made with the alternative of expanding glioma. On March 19, two small trephine openings were made under local anesthesia in the upper temporal and frontal regions on the right side. The dura was exposed and showed a bluish appearance. When opened, a typical subdural hematoma sac was disclosed. When the sac was opened, a large amount of brownish red fluid was evacuated. The sac was irrigated until the irrigation was returned clear. There was no solid clot present. The incisions were closed without drainage. The patient made a very prompt, uneventful recovery.

CASE 6.—H. L., white male, aged 61 years, was admitted to the Church Home and Infirmary on May 20, 1930, and was discharged on June 4, 1930.

The patient gave a history of head injury three months previous to admission to the hospital. While trying to halter a bull, he was thrown against the side of the stall, striking the right side of his head. He was not unconscious, but felt dizzy for ten to fifteen minutes. Following this injury he had intermittent headaches for four to five weeks. The headaches gradually disappeared until three weeks prior to his admission to the hospital, when he suddenly became unconscious while on an automobile trip. He regained consciousness about a half hour later, but remained very drowsy and complained of severe headache until the time of admission. The history obtained from the family showed that his memory had become affected during this three-week period, and at the time he was disoriented as to time and place.

On admission to the hospital, the patient's attention could not be aroused and he was completely disoriented. The neurological examination showed the following positive findings: (1) bilateral choked disks with hemorrhages; (2) exaggerated deep reflexes, left upper and lower extremities; (3) positive Babinski on the left side; and (4) muscle weakness, left upper and lower extremities. A diagnosis of subdural hematoma was suspected, but the possibility of a tumor could not be ruled out. The patient therefore was prepared for a ventriculogram, and under local anesthesia a small trephine opening was made over the posterior horn of the right lateral ventricle. When the dura was exposed, it appeared bluish green in color and when incised there was an escape of dark brownish red fluid. A diagnosis of subdural hematoma was made immediately and the ventriculogram was not done. Under local anesthesia a right subtemporal decompression was done. A portion of the sac of the hematoma was resected and its contents evacuated by irrigation with salt solution, and two small rubber tissue drains were placed in the sac before the incision was closed. The patient drained a considerable amount of dark brownish fluid for several days, after which the drains were removed. He made an uneventful recovery and was discharged from the hospital on June 4, about two weeks after admission.

CASE 7.—J. H., white male, aged 56 years, was admitted to the Mercy Hospital on Dec. 31, 1934, and was discharged on March 1, 1935.

The patient was admitted to the hospital following a head injury sustained in a fall from a ladder. He vomited several times on admission and complained of severe headache. There was a laceration of the scalp in the left parietal region and ecchymosis and swelling of the left upper lid. The neurological examination was entirely negative. X-ray examination of the skull showed a linear fracture in the left frontal region extending into the parietal and occipital bones.

The patient was kept under observation in the hospital for sixteen days. During this period his headaches continued and at times he showed very marked mental confusion and slight elevation of temperature, with a pulse rate ranging between 70 and 90. Spinal puncture on Jan. 5 showed xanthochromic fluid under a pressure of 330 mm. of water. There was no definite change in the patient's condition fol-

lowing the puncture. A diagnosis of subarachnoid hemorrhage was made and several spinal punctures were done during the next ten days. The pressure gradually decreased and on Jan. 15 it was 90 mm. of water, but the fluid had become more xanthochromic, and it was felt that the patient had a clot over the left hemisphere or a subdural hematoma.

On Jan. 16 a left osteoplastic flap was turned down. The external surface of the dura appeared normal, but when an incision was made through the dura there was a free flow of yellow fluid. This fluid was found to be encysted in a sac which was very firmly adherent to the dura. The outer wall of the sac was dark in color and was so tightly adherent to the dura that it had to be removed with the curette. The ventral wall of the sac was made up of thickened pia-arachnoid and the fluid was encysted in an area about 15 cm. in diameter. The sac was only partially removed after its contents had been thoroughly evacuated and the operative field washed with salt solution. The wound was closed without drainage. The patient's convalescence was slow and was marked by symptoms of mental confusion, which were the last to disappear.

CASE 8.—M. McK., colored woman, aged 38 years, was admitted to the Baltimore City Hospitals on Sept. 1, 1935, and discharged on Oct. 8, 1935.

Four days before admission the patient complained of severe headache, became very drowsy, and two days later became irrational. When admitted to the hospital, she was in a semicomatose condition, with a temperature of 102° F., and a pulse rate of 112. There was cervical rigidity, and bilateral choked disks were present. The neurological examination was otherwise negative. The white blood cell count was 17,980. Lumbar puncture showed a clear fluid under a pressure of 250 mm. of water. An encephalogram showed the lateral ventricles markedly displaced to the left beyond the midline, with compression of the right ventricle downward. There was no history of injury obtained and the condition was rather acute for a subdural hematoma. The encephalogram, however, suggested this diagnosis and on Sept. 10 two small trephine openings were made in the right parietal and frontal regions and a subdural hematoma was found. The contents of the sac were evacuated through these two openings and the sac irrigated with salt solution. The incisions were closed without drainage. The patient made an uneventful recovery and was discharged on Oct. 8, 1935.

CASE 9.—M. B., white woman, aged 71 years, was admitted to the Hospital of the Women of Maryland on Feb. 12, 1935, and discharged on March 21, 1935.

One and one-half months before admission to the hospital, the patient tripped and fell down the cellar stairs. There was a small laceration of the scalp and the patient was rendered unconscious for several minutes. She was confined to her bed for ten days following her injury. Her only complaint at this time was pain in the back of the neck. She gradually developed a weakness of the right arm and leg, and one week before admission to the hospital she fell two or three times, due to weakness in the leg. She then developed a severe headache accompanied by mental disturbances and loss of memory. There were also a speech disturbance and incontinence of bowel and bladder. Several days after admission the patient's condition improved somewhat, but she suddenly fell into a comatose state and appeared acutely ill. Lumbar puncture on Feb. 20 showed a xanthochromic fluid under increased intracranial pressure. An encephalogram showed a shifting of the lateral ventricles to the right with a compression of the left ventricle and a slight dilatation of the right.

A diagnosis of subdural hematoma was made. On Feb. 20 under local anesthesia a small trephine opening was made in the left temporal region, and a typical subdural hematoma was found. A second trephine opening was made in the frontal

region and the contents of the subdural hematoma washed out through these two openings. Both incisions were closed without drainage and the patient made a very slow uneventful recovery.

CASE 10.—J. A. R., white man, aged 63 years, was admitted to the West Baltimore General Hospital on Jan. 13, 1932, and died on Feb. 13, 1932.

The patient was admitted three days following an automobile accident in which he was rendered unconscious. He developed headache, pain in the back of the neck, and there was an occasional spell of vomiting. His headache continued until Jan. 19, when he became difficult to arouse. Spinal puncture on Jan. 19 showed a xanthochromic fluid under markedly increased pressure. The patient seemed to improve slightly following the puncture, but the next day he became very drowsy again. He was thought to have a subarachnoid hemorrhage and on Jan. 22 a second spinal puncture was done. At this time the fluid was still xanthochromic, under increased pressure, and contained 11 cells per c. mm. He again improved for a period of six days, at the end of which time he became delirious, attempted to get out of bed, and his temperature and pulse were elevated, and he showed signs of meningitis. A third spinal puncture was done on Feb. 2 with practically the same findings as before. Several days later he developed a slight weakness in his right arm and leg, with increased reflexes on the same side.

On Feb. 5 a small trephine opening in the left temporal region disclosed a subdural hematoma. This trephine opening was gradually enlarged under the temporal muscle and the sac evacuated by washing with salt solution. Two rubber tissue drains were inserted and the wound closed. The patient reacted very well following the operation, recovered the use of his right arm and leg, became cheerful, quiet, and cooperative. Six days following the operation, he had a recurrence of his original symptoms and signs, together with elevation of pulse and temperature. The original incision was reopened on Feb. 12 and the sac explored. There was a collection of greenish yellow fluid in the sac. This was evacuated. A culture from the cystic fluid showed short-chain streptococci. The patient did not react after the second operation and died the following day with streptococcic meningitis.

CASE 11.—A. S., colored man, aged 74 years, was admitted to the University Hospital on Nov. 16, 1933, and discharged on Feb. 3, 1934.

The patient gave a history of falling on a porch and striking his head on a concrete pillar. He was not unconscious at the time and was able to go about his work almost immediately after the fall. Several weeks later he began to have severe headaches accompanied by dizziness and a tendency to fall forward. The patient gradually developed mental changes, became very untidy, and at the time was disoriented.

On admission to the hospital the patient showed a healed laceration over the left eyebrow, early choking of both disks, weakness in the left upper and lower extremities, and a slight left facial weakness. Lumbar puncture showed a clear fluid with a spinal fluid pressure of 300 mm. of water. A chronic subdural hematoma was suspected and on Nov. 28, 1933, a right subtemporal decompression was done, and a hematoma was drained through this incision. The patient showed marked improvement for two weeks following operation. At the end of this time, he suddenly developed signs of increased intracranial pressure and his old symptoms returned. A diagnosis of recurrent subdural hematoma was made, and on Dec. 29 a right sided bone flap was made and the entire subdural hematoma with the sac was removed. His progress following the second operation was slow, but he eventually made a complete recovery and was discharged about a month following his second operation.

CASE 12.—R. P., white man, aged 57 years, was admitted to the University Hospital on March 12, 1934, and discharged on March 24, 1934.

The patient gave a history of a fall on the ice on Feb. 12, 1934, at which time he sustained a fractured skull. He was not unconscious at the time, but about a week following his injury he showed mental disturbances and difficulty in speech. These symptoms gradually improved until March 12, the date of his admission to the hospital, at which time he suddenly became disoriented as to time and place, was aphasic and very emotional. There was a slight weakness in the right arm and leg; the disks showed early choking; and the neurological examination was otherwise negative.

A diagnosis of chronic subdural hematoma was made and on March 14 a left osteoplastic bone flap was turned down and a large subdural hematoma was removed. The patient made an uneventful convalescence and was discharged ten days after operation.

CASE 13.—N. K., white male, aged 47 years, was admitted to the South Baltimore General Hospital on Aug. 1, 1934, and discharged on Aug. 25, 1934.

The patient gave a history of chronic headaches of two weeks' duration, accompanied by dizziness. The neurological examination was negative except for internal strabismus of the right eye. Lumbar puncture showed a xanthochromic fluid. An encephalogram showed a shifting of the lateral ventricles to the left of the midline, with a compression of the right lateral ventricle and a slight dilatation of the left. A diagnosis of a subdural hematoma was suspected and a trephine opening was made over the right hemisphere and the diagnosis verified. The subdural hematoma, together with the sac, was removed through a flap operation on Aug. 13. The patient made a very prompt and uneventful recovery and was discharged twelve days later. In this case the diagnosis was suspected from the encephalogram and was verified by a trephine opening.

CASE 14.—L. W., white woman, aged 63 years, was admitted to the Union Memorial Hospital on June 3, 1930, and discharged on Aug. 18, 1930.

The patient gave a history of having been injured in an automobile accident nine weeks previous to admission. She was unconscious for only a short time. Five weeks later she developed a speech disturbance and weakness of the right arm and leg.

On admission to the hospital there was a small scar over the right temporal region and increase in the reflexes in the right upper and lower extremities, with a positive Babinski on the right side. A diagnosis of subdural hematoma was made and an operation was done on June 5, 1930. Flap operation was the technique used, and a large subdural hematoma was removed. The entire sac was removed. The patient had a stormy convalescence, but at the time of discharge the speech was normal and she had regained function in her right arm and leg.

CONCLUSIONS

1. The symptomatology of chronic subdural hematoma is extremely variable and misleading.

2. Trauma is the etiologic factor, with rare exceptions and is usually considered trivial at the time of injury.

3. The lesion is more frequent in males than in females.

4. The diagnosis should be suspected or made if there is a history of slight injury followed by headache, mental changes, stupor, or signs of slowly increasing intracranial pressure. There may be a "free interval" of weeks, or even months, following the injury before the symptoms appear.

5. Localization is frequently impossible by neurological signs alone. Pathologic reflexes are common, but usually they are of no localizing value.

6. When subdural hematoma is suspected, dual perforations should be made, for the lesion is frequently bilateral.

7. Bilateral trephine openings should be accepted as the method of choice, because this simple procedure serves (a) to establish the diagnosis, (b) to determine whether the lesion is bilateral, and (c) to evacuate the hematoma. When there is a well organized clot present, it may be necessary to reflect a flap to remove it effectually.

8. It is not necessary to remove the sac to effect a cure.

9. Failure of the compressed hemisphere to expand and vascular changes secondary to long-continued compression are the most serious postoperative complications.

I am indebted to Dr. Charles Bagley, Jr., for granting me permission to include several of his own cases in this series.

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SUBDURAL HEMATOMA, ACUTE AND CHRONIC, WITH SOME REMARKS ABOUT TREATMENT

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OF THE entire group of head injuries, those with subdural hematomas most frequently require a critical decision as to whether or not surgical intervention is necessary, and if so what is the most desirable procedure and when is the most favorable time.

The speeding forces of the machine age, particularly the automobile, have increased enormously the incidence of indirect head trauma, and the proper treatment of these seriously injured persons is a growing responsibility.

During the past two decades, our consciousness about chronic subdural hematoma has been sharpened by the publications of Trotter,[1] Putnam and Cushing,[2] and more recently by those of Rand,[3] Jelsma,[4] Gardener,[5] Munro,[6] Coleman,[7] Frazier,[8] Grant,[9] and others, and we are now more alert to suspect the possibility of a chronic subdural hematoma in the differential diagnosis of comatose patients.

The results following the removal of chronic subdural hematomas are uniformly excellent in all neurosurgical clinics. There are indeed few surgical conditions in which early diagnosis and prompt surgical intervention are followed by such dramatic and complete recovery.

Therein, however, lies a danger. Our enthusiasm about the results with chronic subdural hematomas easily may be carried over to the other head injuries with deepening coma.

Whereas operative interference in patients suffering with chronic subdural hematomas is practically always necessary, this is not true of patients with acute subdural hematomas. Hasty operation on such patients can only end disastrously. The injured person with only a small margin of safety who might well be saved by medical measures may easily have his chances jeopardized by manipulations incident to taking of x-ray pictures, transportation to the operating room, and the added trauma of the operation.*

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*Patients in shock, whether it be the result of trauma, a prolonged and exhausting operation, or an unusual response to some medication, have a small margin of safety which is best reflected by temperature, pulse, and blood pressure readings. Exposure to changing temperatures or undue handling are factors which aggravate surgical shock. Studies of the influence of movement on shock have recently been reported by Miller,[10] who recommends the bringing of the bed to the operating room, a custom practiced in Cushing's clinic for many years.

There is need, therefore, for a clearer differentiation of subdural hematomas into the acute and chronic phases. Selection of patients with acute subdural hematomas who can be benefited by operation is of utmost importance.

ACUTE SUBDURAL HEMATOMA

Acute subdural hematoma is an accompaniment of severe head trauma in which, as a result of injury to cerebral vessels, there has been bleeding into the subdural space. Of and by itself, acute subdural hematoma can hardly be said to exist, for invariably there is associated serious if not extensive laceration of the brain as well as a fracture of the skull. The bleeding is arterial as well as venous. From the injured vessels varying quantities of blood spread through the subdural space and the adjoining subarachnoid meshes. Often a fine layer of blood dissects its way beneath the pia mater.

A brief consideration of the mechanics involved in this type of injury will aid in the better understanding of the relative effects upon the brain and skull following a transmitted blow to the head. Because of the suspended position of the brain, a transmitted injury to the head will cause the brain to travel at a different rate of speed than the skull. At the moment of impact, the skull starts to move ahead of the brain which, because of its inertia, lags behind. The brain, having started to move later, still continues to be propelled after the moving force of the skull has spent itself. It is during this period of shift that the brain, continuing in its course, is pitched against the jagged prominences at the base of the skull with resulting cerebral laceration and vascular rupture. As one would expect, the most frequent sites of damage are at the tips of the temporal and frontal lobes.

Patients with acute traumatic subdural bleeding present a clinical picture varying with the degree of brain laceration and the associated intracranial bleeding. A single and small laceration of the brain is accompanied by moderate bleeding into the subdural and subarachnoid spaces with evidence of pinkish fluid by lumbar puncture. These patients as a rule are immediately unconscious following the injury and within several hours begin to show signs of recovery. Consciousness may return within an hour or twenty-four hours, depending upon the severity of the head injury. Neurological signs are few and inconstant. The best guide to the degree of cerebral trauma is the varying state of the patient's consciousness. Signs of returning consciousness within several hours point to a favorable outcome and are far more reliable guides of the patient's condition than the pulse or blood pressure readings. Increasing stupor and coma after an interval of twenty-four hours usually means extensive brain laceration, cerebral edema, and massive bleeding. The outlook under such circumstances is unfavorable.

Excluding compound and depressed fractures of the skull and extradural bleeding, acute intracranial injuries are best treated medically by absolute rest, by measures combating shock, by moderate dehydration, and by the judicious use of lumbar puncture. That conservative management of acute head injuries is most desirable was very conclusively brought forth by Wortis and Kennedy[11] in their study of 1,000 consecutive cases of head injury at Bellevue Hospital. There can be but little doubt that patients with mild cerebral laceration and small quantities of blood in the subdural space will respond best to the medical measures mentioned. Only harm can come to these patients by operative manipulations. Those who have sustained severe brain laceration with resulting extensive subdural collections of blood seldom are helped by operation and the removal of blood from the subdural space rarely alters the course of events. Thus, of the 257 cases that came to autopsy in the series reported by Wortis and Kennedy,[11] 96.9 per cent revealed a fracture of the skull and 91.8 per cent showed brain laceration.

There are, however, a few cases, rare to be sure, with moderate laceration of the brain in which recovery might well ensue if the associated acute subdural hematoma is removed. When such patients recover, they are usually left with disabilities depending upon the extent of the brain damage.

Of several hundred cases of acute subdural hematoma that have come under my observation at Bellevue Hospital, there were only four in which operation seemed to offer some hope. With one exception, practically no improvement was noted following the removal of the acute subdural hematoma. The autopsy specimens disclosed such extensive and irreparable brain damage that one could not hope for anything but a fatality. The one patient (Case 3) who fortunately and surprisingly recovered was left with a paresis of the left arm and a left homonymous hemianopsia. The progression of symptoms after an interval of improvement following the trauma and the focal nature of the convulsions were the indications for operative intervention in this patient.

The history and findings in the four operative cases of acute subdural hematoma follow:

CASE REPORTS

CASE 1.—Acute alcoholism, head trauma, stupor upon admission. External evidence of severe injury, bloody spinal fluid. Improvement for one week then return of stupor, left-sided weakness, dilated right pupil. Spinal fluid xanthochromic. Bilateral trephine, bilateral hematoma without capsule formation. Fatality. Autopsy revealed extensive brain laceration.

History: Thomas W., an unidentified man, aged 56 years, was brought to the hospital on Nov. 26, 1935, in a confused and stuporous condition. He evidently had been in a fight while drunk.

Examination: The patient was unable to cooperate. There was a ragged scalp laceration over the left parietal region, ecchymosis about the right eye, clotted blood in both nostrils, and laceration of the lower lip. Except for depressed reflexes, there were no abnormal neurological signs.

Laboratory data: Temperature, 99; pulse, 84; blood pressure, systolic 130, diastolic 74. The spinal fluid was uniformly bloody.

Course: There was very little change in his condition until three days later when he began to show signs of returning consciousness. After three days, he again became stuporous. The right pupil was larger than the left; the neck was stiff; fundi showed no abnormality. A suggestive hemiparesis was present on the left and Babinski sign was positive on both sides. The spinal fluid was xanthochromic and the pressure apparently normal. He was given hypertonic intravenous glucose and caffeine sodium benzoate, but he failed to show any improvement.

Operation: Local novocain anesthesia. Trepanation over the right postparietal region revealed greenish colored dura. When the dura was opened, there was a gush of dark brown fluid. There was no evidence of a membrane. About 30 c.c. of old blood was aspirated, and irrigations with warm normal saline were continued until the returning fluid was clear.

Trepanation then was performed over the left postparietal area. Here also the dura appeared greenish. When the dura was opened, there was no evidence of any membrane. About 15 c.c. of old unclotted blood was aspirated. Irrigations were carried out as on the right side.

Postoperative course: The patient failed to show any signs of improvement. The following morning he died.

Autopsy: The brain showed multiple areas of laceration. The right temporal lobe particularly was markedly softened and contained a large intracerebral clot. (Fig. 1.)

Comment.—With such extensive brain damage, the patient usually is in profound shock and it is not surprising to find very few localizing signs. Our experience has shown that capsule formation seldom takes place where there has been cortical laceration. Operative intervention in this patient seemed advisable for the following reasons: Because following the initial improvement there was a relapse with the appearance of definite localizing signs pointing to the formation of a hematoma. In favor of this conception was also the change in the character of the spinal fluid from a uniformly bloody color to a xanthochromia. It was hoped that the cerebral laceration would be minimal and that the removal of the acute hematoma would permit a satisfactory cerebral compensation. The autopsy findings, however, revealed cerebral lacerations far too extensive for surgical relief.

Case 2.—Direct head injury with hammer, no loss of consciousness, headache, vomiting, and lethargy for six days. Drift of right arm, Babinski on left, bloody spinal fluid under increased pressure, x-ray of skull negative, progressive right hemiparesis with early papilledema, bilateral trepanation, removal of subdural blood and clots. Immediate improvement. Fatality four days later. Autopsy revealing basilar fracture, subdural hemorrhages, and cerebral edema.

History: Harry B., a 53-year-old janitor, was brought to the hospital on Nov. 5, 1935, because of headaches and drowsiness. Six days before admission, while working in the furnace room, a hammer fell and struck him over the left parietal

region. He was momentarily stunned, fell to the floor, but was able to pick himself up and ascend the stairs. However, he had to be helped into the house. That night and the following day he continued to complain of headache and vomited repeatedly. The headaches persisted; he continued to vomit and gradually became more somnolent.

Examination: The patient was drowsy, but he could be aroused, at which time he was fairly well oriented and complained of generalized headaches. There were abrasions over the left parietal and right occipital regions with adjacent areas of ecchymosis. The pupils were slightly irregular but reacted well to light and accommodation. The fundi showed no abnormality. There was no facial weakness. When both arms were outstretched, there was a drift of the right arm. Response to pinprick was equal on both sides. The deep reflexes were equally sluggish. Babinski sign was present on the left, but doubtful on the right. Blood pressure was systolic 140, diastolic 88.

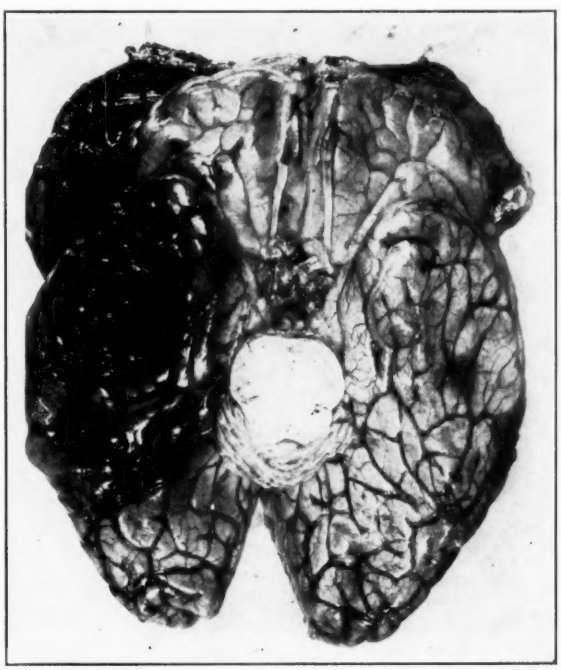

Fig. 1 (Case 1).—Acute subdural hematoma with multiple cerebral lacerations, the most extensive in the right temporal lobe.

Laboratory data: The urine showed no abnormality. Hemoglobin, 88 per cent; red blood cells numbered 4,650,000 per c.mm.; leucocytes were 15,450 per c.mm., with 76 per cent polynuclear forms and 21 per cent lymphocytes; Kline test was negative; blood sugar, 103 mg. per cent; urea nitrogen, 11.2 mg. per cent.

X-ray of the skull gave no evidence of a fracture. The spinal fluid was uniformly bloody, under 330 mm. of pressure. After the removal of 20 c.c., the pressure was reduced to 100 mm.

Course: During three days of observation, the pulse fluctuated between 50 and 60 per minute. At times the patient was alert, at other times he was hard to awaken. Weakness of the right side of the face became evident, the fundi began to show early papilledema, and the right-sided weakness progressed. Spinal fluid was blood tinged under 230 mm. of pressure; after the removal of 10 c.c. of fluid, the pressure was reduced to 100 mm.

Operation: On Nov. 8, 1935, under local novocain anesthesia, a bilateral trepanation was performed over the parietal regions. Through the left trephine hole about 15 c.c. of fresh blood and some clots were removed from beneath the dura. The brain appeared congested and edematous, but no gross blood was found in the subdural space.

Postoperative course: On the following day he was more alert, motion in the right arm and leg improved, the deep reflexes responded well on both sides, but the Babinski sign on the left persisted. Twenty-four hours later he was more drowsy and the right-sided paresis returned. Spinal fluid was xanthochromic, showing a pressure of 420 mm. He gradually became more comatose and died four days after operation.

Autopsy disclosed a fracture of the base of the skull through the left petrous bone, subdural hemorrhages, and marked edema of the left temporal lobe.

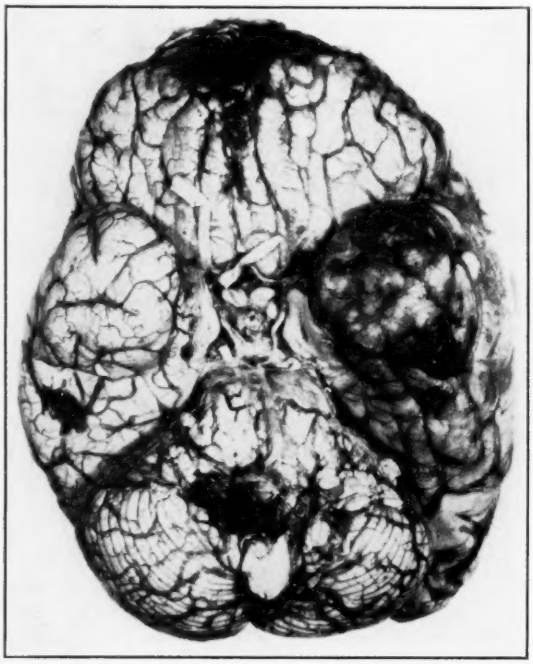

Fig. 2 (Case 2).—Multiple cerebral lacerations most marked at frontal and temporal lobes.

Comment.—Unquestionably the direct blow to the head was much more severe than was suspected upon first examination. The uniformly bloody spinal fluid under such increased pressure pointed to serious intracranial injury. The progressive somnolence and the advancing focal signs over a period of nine days were sufficient indications for operation. However, the autopsy (Figs. 2 and 3) proved that the extensive fracture traversing the left petrous bone, the cerebral hemorrhage, and edema were beyond surgical help.

Case 3.—Injury to forehead during alcoholic spree. Left-sided convulsions, lethargy, and confusion. Left hemiparesis and hemihypesthesia. Spinal fluid xanthochromic. Bilateral trepanation, acute subdural hematoma on right associated with brain laceration. Recovery with residual spastic paresis of left arm

and left homonymous hemianopsia. Recurrence of left-sided Jacksonian seizures three months later. Encephalograms revealed dilatation and retraction of right lateral ventricle as a result of scarring. Luminal therapy. No seizures for over a year.

History: Mary D., a 49-year-old housewife, was admitted to the hospital in a state of confusion. Seven years previously she had been treated in the psychopathic division for chronic alcoholism.

The patient had been drinking to excess for some time, but one week before admission, while trying to get out of bed, she had bumped her forehead against the dresser. There was no loss of consciousness. Three days later she had a convulsion involving the left arm and left leg. She became drowsy, confused, and was brought to the hospital.

Shortly after admission she had two convulsions. The head and eyes turned to the left, the left side of the face was drawn up, and the left arm and left leg were in tonic spasm.

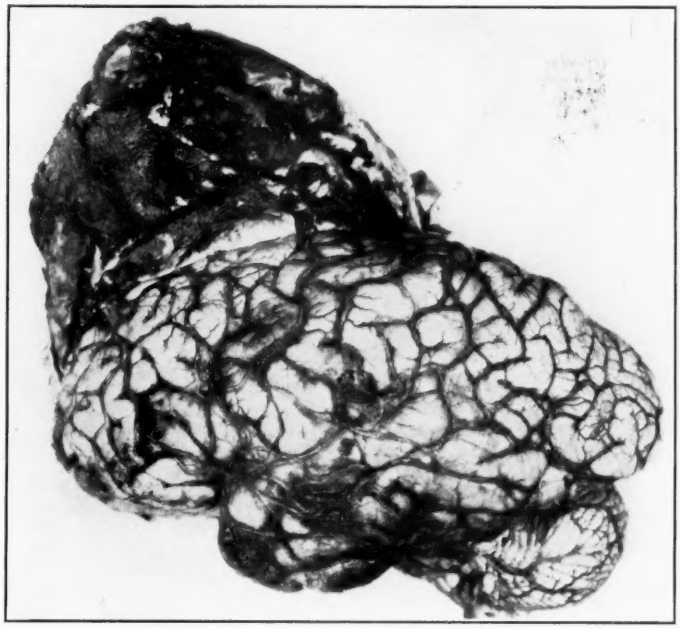

Fig. 3 (Case 2).—Acute subdural hematoma secondary to multiple cerebral lacerations.

Examination: The patient was drowsy but could be aroused sufficiently to state that she had right-sided headache. There were numerous areas of ecchymoses all over her body. The pupils were under the influence of homatropine. The fundi had a frosted appearance, but there was no papilledema. Ocular movements were normal in all directions. There was a distinct left central facial weakness, and the left hemiparesis as well as the left hemihypesthesia involved the arm more than the leg. The deep reflexes were more active on the left, though the ankle jerks could not be obtained on either side. The abdominal responses were absent on both sides. Babinski sign was positive on the left.

Laboratory data: Temperature, 101.4; pulse, 114. Urine showed two plus albumin but was otherwise negative. Blood pressure: systolic, 100; diastolic, 70. Red cells numbered 3,750,000 per c.mm. Leucocytes, 6,100; 75 per cent polynuclear cells, 22 per cent lymphocytes; hemoglobin, 70 per cent; N.P.N., 40. Spinal fluid was xanthochromic. Wassermann was negative.

Course: After four days of observation, during which period she gradually became more comatose, it seemed imperative that the presence of a subdural hematoma be eliminated.

Operation: Oct. 10, 1935, under local novocain anesthesia, bilateral trepanation was performed over both postparietal regions. The dura on the left appeared normal but when it was incised about 2 c.c. of brownish fluid escaped. The underlying brain appeared congested. The dura on the right was dull gray and under tension and when opened 30 c.c. of dark brown fluid was aspirated. The cranial defect was enlarged to about the size of a half dollar, and with irrigations through a catheter about two ounces of bluish black blood clots were washed out. When the return washings were clear, the exposed brain appeared congested and there were several areas of ecchymoses and laceration. There was no evidence of any capsule.

Fig. 4 (Case 3).—Dilatation and retraction of right ventricle suggestive of cerebral atrophy and scarring.

The sunken brain began to pulsate freely and rise to the surface as more and more of the old blood and clot was removed. During one period of the irrigation, the blood pressure suddenly rose to 200 systolic and within a few minutes returned to the preoperative level of 120 systolic, 90 diastolic.

Postoperative course: The next day she was still drowsy but was taking fluids. The left leg moved more freely, but the left arm remained spastic. Spinal fluid was uniformly pink under extremely low pressure. During the first postoperative week she was given daily lumbar punctures and hypertonic intravenous glucose. She became more alert, motion in the fingers of the left hand began to return, but she had extreme nausea for several days. Her improvement was slow. She was discharged twenty-four days after the operation at which time she was mentally clear, could walk without assistance, but still had weakness and spasticity of the left arm. Visual fields showed an almost complete left homonymous hemianopsia.

Second admission: On Feb. 19, 1936, she was readmitted to the hospital because of increasing left-sided Jacksonian seizures. She had been well for two months after leaving the hospital and then noted recurrent twitchings of the left arm and left side of the face.

Examination: The fundi were normal except for moderate retinal sclerosis. Vision O. D. 20/200, O. S. 20/200. The pupils were Argyll Robertson in type. There was a slight left facial weakness, a spastic left hemiparesis particularly involving the left arm, diminished left corneal reflex, overactive deep reflexes on the left, Hoffmann sign on the left, a left hemihypesthesia, and left homonymous hemianopsia. Blood pressure: systolic, 180; diastolic, 108. Blood Wassermann negative.

Encephalograms: On Feb. 28, 1936, after the removal of 110 c.c. of clear spinal fluid, 120 c.c. of oxygen was injected. X-ray films revealed considerable dilatation of the right lateral ventricle with retraction to the right (Fig. 4). The third ventricle was visualized and appeared slightly dilated. The finding suggested cerebral atrophy and scarring.

The patient was put on luminal therapy and has been free from seizures since.

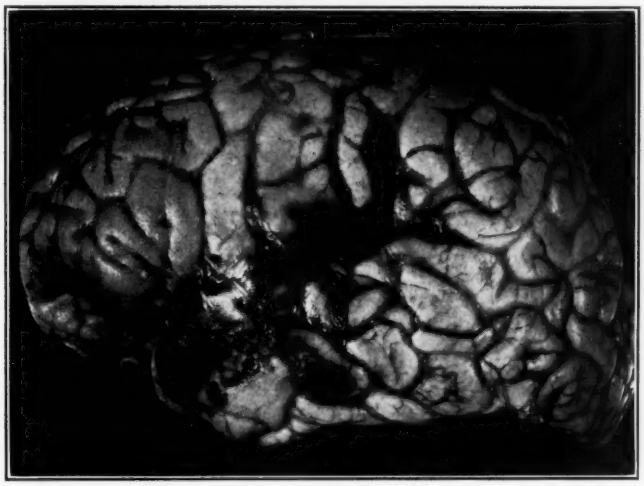

Fig. 5 (Case 4).—Multiple cerebral lacerations associated with acute subdural hematoma.

Comment.—The preoperative diagnosis that there was cerebral cortical laceration in association with subdural bleeding was made because of the history of convulsions and the widespread motor and sensory disturbance. It should be noted that there was no evidence of a capsule. The patient's recovery was unexpected because of the extensive cerebral damage that was found. The residual paresis of the arm was the only incapacity. The encephalographic findings upon her second admission show an interesting late result of cerebral damage.

CASE 4.—Acute head injury, coma, Cheyne-Stokes respiration, generalized convulsion, right hemiparesis, bloody spinal fluid, left-sided convulsion, left hemiparesis. Negative bilateral trephine exploration. Autopsy showed fracture base of skull with extensive cerebral laceration.

History: On Sept. 27, 1936, Robert D., a middle-aged unknown white man was picked up on the street in a comatose condition and brought to the hospital by ambulance. En route he vomited and had a generalized clonic convulsion.

Examination: The patient presented the picture of decerebrate rigidity. Respirations were Cheyne-Stokes in type; pulse was 72 per minute. There was some ecchymosis about the right eye, and further observation disclosed that he had a right facial weakness and a right hemiparesis.

Laboratory data: The urine revealed no abnormality. The spinal fluid was uniformly bloody, under 240 mm. of water pressure.

Course: That evening he had a left-sided convulsion with spasticity and cogwheel rigidity of the left arm. The deep reflexes on the left were hyperactive and the Babinski sign and ankle clonus were positive on the left. The next day there was spasticity of all the extremities, bilateral positive Babinski, and the spinal fluid was uniformly bloody, under 350 mm. of water pressure. The temperature rose to 105.6 and respirations were 40 per minute. However, within the next two days his condition improved surprisingly. The right pupil became dilated, but the left hemiparesis persisted.

Operation: Sept. 30, 1936, under local novocain anesthesia, a bilateral trephine was performed over both postparietal regions. No subdural clot was found on the left. On the right about 25 c.c. of pink fluid escaped from the subdural space.

Postoperative course: During the ensuing week he improved slowly, but then became confused and disoriented. He died twelve days after the trephine exploration.

Autopsy revealed fracture of the base of the skull in the middle fossa, laceration of the under surface of the left temporal lobe (Fig. 5), some blood-stained fluid in the subdural space on both sides, and a bilateral bronchopneumonia.

Comment.—Because he showed signs of improvement during the first three days after admission, it seemed worth while to give him the benefit of a bilateral trephine exploration. The autopsy findings revealed that the cerebral damage was beyond surgical aid.

It is well to realize, therefore, that in the great majority of patients suspected of having an acute subdural hematoma, operation should be withheld. The mild cases will improve under a medical regime. Those severely affected cannot be helped by operative decompression. There are, however, a small number of patients who, after an interval of improvement, begin to show signs of increasing stupor with evidence pointing to a localized collection of blood. Upon these patients operation should be performed.

CHRONIC SUBDURAL HEMATOMA

In a previous publication[12] it was pointed out that chronic subdural hematoma occurs more frequently than middle meningeal hemorrhage. Also, clinical and experimental evidence was presented to prove that a chronic subdural hematoma is the result of a relatively mild trauma to the head which displaces the brain in an anteroposterior direction with a resulting tear of one or more of the cerebral veins as they enter the superior longitudinal sinus. The vein, being least supported as it traverses the subdural space, yields at this point. The venous bleed-

ing that follows into the subdural space is slow, intermittent, and variable and will be influenced by changes in the intracranial tension.

Within ten days to a fortnight, a fine membrane, arising chiefly from the inner lining of the dura, encloses the blood and gradually a cyst wall is formed.

When the intracranial tension falls below the venous pressure, there is a repetition of venous oozing into the cyst until the tension within the cyst wall is sufficient to prevent any further venous bleeding. Headache, vomiting, and lethargy are the most constant symptoms during this phase. As the clot within the cyst retracts and partial organization takes place, the intracranial tension readjusts itself and the patient becomes more alert and at times fully oriented.

The variations in tension within the cyst not only account for the fluctuations in consciousness but also explain the inconstant state of the pupil, the changing paresis, the appearance and disappearance of facial weakness, and Babinski sign. It is not unusual to find the hemiparesis, the dilated pupil, and the chronic subdural hematoma all on the same side.

If one were to make a composite of the clinical stories and findings in this series of fifteen patients with chronic subdural hematoma, it would read as follows:

A middle-aged or elderly individual receives a mild injury to the frontal or occipital region which may be totally forgotten. There is a latent interval from two days to six weeks from the time of injury to the onset of headache. The headache is intermittent and is by far the most constant symptom. Soon lethargy, somnolence, vomiting, mental changes, diplopia, and dizziness follow. Paresis of one side of the body often associated with a dilated pupil on that side may be the only neurological signs in addition to a central facial weakness. The patient becomes gradually more lethargic and then comatose. The symptomatology and findings are often so inconsistent and fluctuate so frequently that one is easily confused. And yet these fluctuations are most characteristic in a patient with a chronic subdural hematoma. These patients are often suspected of a cerebral neoplasm, posttraumatic neurosis, encephalitis, cerebral arteriosclerosis, cerebral thrombosis, and psychoneurosis.

The spinal fluid is usually under increased pressure, and often xanthochromic, but it may be perfectly clear and colorless.

Many a time the only certain and safe procedure to establish the diagnosis is to perform bilateral trepanation.

The history and findings in the fifteen operative cases of chronic subdural hematoma follow.

CASE 5.—No history of head trauma. Headache, diplopia for one month. Period of unconsciousness. Increased spinal fluid pressure and early papilledema. Slight

mental retardation. Inequality of pupils, bilateral external rectus palsy, advancing papilledema, right facial weakness, and bilateral Babinski. Spinal fluid pressure 280 mm. Palsy left side of body under observation. Chronic subdural hematoma over left hemisphere. Removal by craniotomy. Recovery.

History: Fred D., an Italian college student, aged 19 years, entered the hospital on Oct. 1, 1933, complaining of headaches of three weeks' duration. He was perfectly well until one month before admission when he experienced severe frontal headache while drilling in the hot sun in the south. The following morning he was sufficiently well to return to his regular duties, but four days later, during drill, the headache returned and persisted. It was associated with the vomiting of medication on two separate occasions. He was taken to the college infirmary where on the second day he became unconscious and fell out of bed. Upon regaining consciousness he began to have generalized dull headache and then to see double. He also noted numbness of the fingers of the left hand. Because of some stiffness of the neck, a lumbar puncture was done which showed normal fluid under markedly increased pressure, and following this procedure his headache diminished. Vision in the right eye, however, soon beegan to fail. He noted tingling sensations in the face and both hands and an early papilledema was observed on the right. Lumbar puncture was repeated with complete disappearance of headache, but this had no influence on the advancing papilledema. There was no somnolence, dizzy spells, convulsions, or disturbance of speech. His mental processes seemed to be slightly retarded. He was sent to the hospital with a diagnosis of cerebral neoplasm.

Examination: The patient was an alert cooperative young man who gave a clear and consistent account of his illness. The positive neurological findings were: (1) the right pupil slightly larger than the left, (2) bilateral papilledema, 4 diopters elevation on the left, and 3 diopters elevation on the right, (3) bilateral external rectus paralysis, (4) slight right facial weakness, (5) stiffness of the neck and bilateral Kernig sign, (6) depressed deep reflexes, (7) active abdominal responses, and (8) bilateral Babinski sign.

Laboratory findings: Urine and blood studies were negative. The blood pressure was 136 systolic and 90 diastolic. Cerebrospinal fluid was clear, showed a pressure of 280 mm. with five cells and a negative Wassermann reaction.

Course: Repeated questioning about the fall out of bed did not convince the staff of its etiologic relationship to the illness, and the service was inclined toward the diagnosis of a neoplasm, either of the posterior fossa or in the brain stem.

The patient's condition remained unchanged for ten days. He then began to show weakness of the left leg and the following day weakness of the left arm. In addition there was subjective numbness of these parts but no sensory disturbance could be established. Ventriculograms were undertaaken for the purpose of localization and it was during this procedure that a hematoma was disclosed.

Operation: On Oct. 1, 1933, a trephine over the left hemisphere brought to view a grayish dura under moderate tension. When the dura was opened, there was a profuse gush of dark brownish blood, 30 c.c. in all, which spurted with each pulsation of the brain. Upon further investigation some shaggy membrane was removed through the trephine hole which established the diagnosis of chronic subdural hematoma. During this procedure it was noted that the left external rectus paralysis completely disappeared. Knowing the frequency with which this lesion is bilateral, another trephine hole was made over the right hemisphere. The dura and the underlying brain appeared normal.

Two days later the patient's temperature rose. He spat some blood-streaked sputum and for thirteen days was ill with a mild form of bronchopneumonia. The left hemiparesis disappeared within five days after the trephine exploration.

On Nov. 14, 1933, craniotomy was performed and a large flap was turned down on the left side, exposing a well-organized hematoma covering the entire left hemisphere. It was grayish brown, lens shaped, tapering off to meet the arachnoid at the periphery, and firmly adherent to the dura but not to the arachnoid. It was so large that it had to be removed in three pieces (Fig. 6). As one-half of the hematoma was raised, a cortical vein could be seen running into the under surface of the hematoma, probably the source of the venous bleeding. This was clamped and severed.

On the day following the craniotomy there was a mild sensory aphasia, and on the next day he suddenly complained of headache, became more aphasic and drowsy, and developed weakness of the right arm. Several ounces of dark blood were aspirated from beneath the flap resulting in prompt improvement in the above signs. Dakin tubes were left in place for the escape of further bleeding. Several days later he showed evidence of wound infection which was fortunately brought under control.

Upon discharge on Dec. 12, 1933, he was completely well.

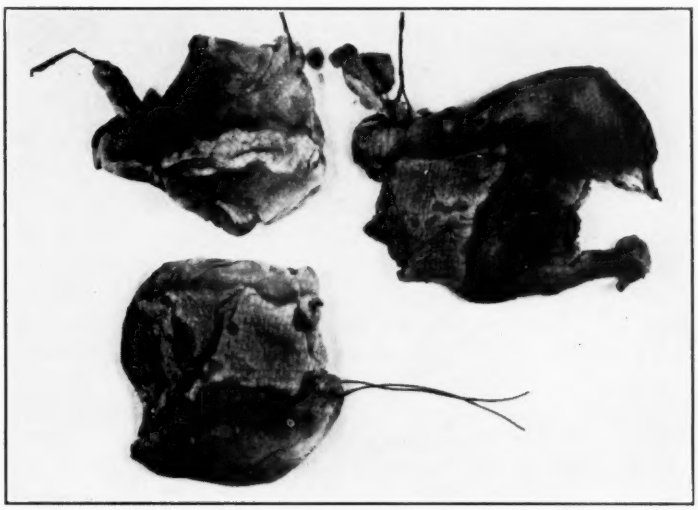

Fig. 6 (Case 5).—Extensive chronic subdural hematoma covering almost entire left hemisphere. Removal through craniotomy wound in three portions.

Comment.—Although there is no history of head trauma preceding the onset of symptoms, he may very well have sustained an injury as he fell out of bed. It is surprising that a lesion so extensive should fail to produce aphasia or a hemianopsia. It is also to be noted that the paresis, though mild, was on the same side of the lesion. This patient is the only case in which a craniotomy was performed after drainage through a trephine hole proved unsuccessful. The finding proved that such a well-organized hematoma can only be properly removed by a craniotomy.

CASE 6.—Story of head injury indefinite until after operation. Headaches, dizziness, and somnolence for two weeks. Occasional vomiting and disturbance in gait. Semistuporous state, right facial weakness, left Babinski, spinal fluid lemon yellow tint and under increased pressure. Bilateral trepanation, subdural hematoma on left evacuated. Uneventful recovery.

History: Frank S., a 57-year-old cook, was brought to the hospital in a somnolent condition. The ambulance surgeon obtained an indefinite story from a friend that the patient had had some accident three weeks previously. The details of the accident were not known. For a week after the accident, the patient was able to work, then he began to complain of headache and dizziness. These gradually increased; he became lethargic and vomited on several occasions. He soon found difficulty in walking.

During a period of four days' observation at the hospital, his state of consciousness fluctuated considerably every few hours. When he was more alert, he complained of headache over the right frontal region.

Examination: The patient was semistuporous, would not talk or respond to commands, but could be aroused by painful stimulation. The peripheral vessels showed moderate arteriosclerosis. Blood pressure: 130, systolic; 70, diastolic; pulse, 68 per minute. There was some stiffness of the neck but no Kernig reaction. Pupils were equal, reacted well, and the fundi showed no abnormality. A right facial weakness was questionable. Both arms seemed to have cogwheel rigidity, but there was no paresis. Sensation could not be accurately tested. Babinski sign was positive on the left and occasionally was found on the right. Abdominal responses could not be elicited.

Laboratory data: The urine was negative. Spinal fluid had a slight lemon yellow tint and was under 250 mm. pressure. There were only 3 lymphocytes per c.mm. Total protein 55 mg. per cent.

Operation: Aug. 29, 1936, under local novocain anesthesia, bilateral trepanation was performed over both postparietal regions. The exposed dura through the right trephine hole was under tension and not discolored. The underlying brain was edematous but otherwise appeared normal.

The dura on the left, however, was slightly bluish. When it was incised, typical dark chocolate colored fluid of a hematoma began to flow. The cranial defect was enlarged to the size of a half dollar. About 180 c.c. of such fluid was obtained from the subdural space. No membrane was seen. Irrigations of warm saline were directed through a catheter to various portions over the left hemisphere until the return washings became clear. The brain was seen sunken about an inch below the surface of the dura. As the fluid escaped from the subdural space, the patient became alert, responded to simple commands, and started to speak. With repeated irrigations the cerebral cortex approached the surface of the dura.

Postoperative course: On the day following the operation the patient was alert, rational, and very cheerful. He volunteered the story that about two and one-half weeks previously he tripped on an orange peel and fell, striking his head against the pavement. He believes he was unconscious for one-half an hour. However, he returned to his quarters where he slept a few hours and then awoke with intense headache. This increased; he felt dull and sleepy, and could not eat. He lost consciousness sixteen days after the injury and was brought to the hospital.

His postoperative course was remarkably smooth. On the seventeenth day after the operation, he was discharged symptom free with no abnormal neurologic findings.

Comment.—A reliable history of head trauma may not be obtained until after the evacuation of a subdural hematoma. In doubtful cases it is far better to explore through a bilateral trephine which, under novocain anesthesia, is simple and not in the least shocking. The regrets have usually been in failing to carry out such a procedure.

The localizing signs indeed were very few and conflicting, in spite of the presence of 180 c.c. of fluid. There were no suggestions of any aphasic components either before or after operation.

The membrane enclosing the subdural fluid must have been so fine that it was not detected at operation, for otherwise the spinal fluid would have shown more than a slight lemon yellow discoloration.

CASE 7.—Trauma to left frontal region. Latent interval six days. Headache and coma of acute onset. Dilated right pupil, right facial weakness, right spastic hemiparesis, clear spinal fluid. Bilateral trephine. Chronic subdural hematoma on right. Uneventful recovery.

History: Charles R., an unemployed lithographer, aged 49 years, was brought to the hospital on April 10, 1934, in a comatose state. The fragmentary history which was volunteered by his landlady seemed to establish the facts that one week before admission the patient received a blow over the left forehead during a brawl. He did not lose consciousness. Six days after the injury he complained of some headache, felt drowsy, and then lapsed into coma.

Examination: The patient was stuporous, but with sufficient stimulation could be slightly aroused, when he would yawn repeatedly. He was incontinent. Fading ecchymosis was observed about the left eye and left temporal region, and the lacerations over the left forehead were completely healed. There were also areas of ecchymosis about the left elbow and left hand. The vessels in the fundi were congested, but there was no papilledema. The pupils were slightly irregular, the right larger than the left.

There was a right facial weakness. Although all the extremities were somewhat rigid, the spastic hemiparesis on the right was very distinct. The deep reflexes were very active, more so on the right. Hoffmann, Gordon, Oppenheim, and Babinski signs were positive on the right. The Babinski sign on the left was doubtful.

Laboratory data: The spinal fluid was clear, colorless, under normal pressure, showing only 9 cells per c.mm.

Preoperative impression: We suspected that this patient had a chronic subdural hematoma on the right, but, because of the injury to the left temporal region, the possibility of an extradural hemorrhage on the left was also considered.

Operation: April 11, 1934, under local novocain anesthesia, a trephine was performed over the right postparietal region. The exposed dura had a greenish tint, and a bluish discoloration could be seen shining through. When the dura was opened, a bluish cystic membrane came to view. This was opened and about 200 c.c. of muddy dark brown fluid was aspirated. Irrigation with warm normal saline was performed in all directions with a No. 14 catheter until the return fluid was clear.

A trephine hole was then made over the left temporal region to rule out an extradural as well as a subdural clot. The dura was normal in appearance, and the underlying brain appeared natural.

By the time the dressing was applied, the patient had regained consciousness, the pupils were equal, the spasticity of the right side of the body had practically disappeared, and the patient carried on an intelligent conversation.

Course: On the second postoperative day he was sitting up reading a newspaper. His convalescence was smooth and without incident. A fortnight after the operation he was discharged, showing no abnormal neurological signs.

Comment.—The dilated pupil, the hemiparesis, the facial weakness, and the Babinski sign were all homolateral with the hematoma. This is not an unusual experience. In fact, it is so often the rule that we

have come to rely upon the dilated pupil as a localizing sign in preference to the hemiparesis. Nevertheless a bilateral trepanation is the operation of choice for in about 10 per cent of cases the hematoma is bilateral. In this patient we have a good example that clear spinal fluid, not under increased pressure, is consistent with the presence of a chronic subdural hematoma.

CASE 8.—Head injury without loss of consciousness. Latent interval two weeks, under observation, appearance of mental changes, somnolence, dilated left pupil, right spastic paresis. Clear cerebro-spinal fluid, coma and fever. Bilateral trepanation. Encapsulated subdural hematoma on left. Meningeal signs and left sided weakness one week after operation. Repeated lumbar puncture followed by complete recovery.

History: Joseph F., a lawyer, aged 47 years, was brought to the hospital on July 6, 1934, in a somnolent state. One month previously he was "held up" and beaten over the left side of the face and forehead, but he did not lose consciousness. In spite of frontal headaches, he managed to attend to business for about two weeks, when it was noted that at times he was disoriented, and soon he began to limp. Shortly thereafter the right arm and right leg became somewhat stiff and weak. Patient was right handed. There were no convulsions or aphasia.

Examination: The patient was very drowsy. When aroused he was confused, disoriented, and uncooperative. There was slight ptosis of the right lid. Left pupil was larger than right. Fundi were normal. Palsy of the right external rectus was slight. There was definite paresis and some spasticity of the right arm and leg. The neck was rigid. Babinski was positive on the right but doubtful on the left.

Laboratory findings: Urine and blood studies were negative. Blood pressure was 145 systolic and 98 diastolic. Cerebrospinal fluid was clear and colorless with only 3 lymphocytes per c.mm.; pressure was 120 mm. Wassermann test was negative.

Course: On the second day following his admission, the temperature rose to 104, pulse was 135, and respirations were 25 per minute. He became more comatose and could not be aroused.

Operation: July 8, 1934, under local novocain anesthesia, a bilateral trepanation was performed over the postparietal regions. When the dura was exposed on the left, it appeared dull gray but had none of the greenish tint usually found. When the dura was opened, there came into view a dark brown shaggy membrane. This membrane was pierced and 150 c.c. of old brownish blood was aspirated. Through a No. 14 catheter irrigation with warm normal saline solution was performed in all directions until the return of fluid was clear. When the hematoma was evacuated, the brain was seen about 3 cm. below the surface of the dura but with repeated irrigation the brain began to pulsate and rise to the surface.

The trephine hole on the right exposed dura normal in appearance. The dura was opened but there was no evidence of a hematoma.

Postoperative course: On the day following the operation, the patient was conscious though irritable and difficult to manage. Temperature, 100.2; pulse, 70; pupils were equal. There was still some spasticity on the right side of the body. His progress was very satisfactory for about a week. When the temperature rose to 104, he became disoriented, his neck became stiff, and some weakness of the left arm was noted. Lumbar puncture was performed and 35 c.c. of blood tinged fluid was removed. His condition improved, lumbar puncture was repeated, and a like quantity of spinal fluid was removed. The fluid was xanthochromic. His condition

from then on steadily improved. Power in the left arm returned. He became fully oriented and spoke very clearly. On the nineteenth postoperative day he was discharged, symptom free, and had no abnormal neurological signs.

Comment.—A trauma to the frontal region, not sufficient to cause unconsciousness, was followed by a latent period of two weeks. Again it was surprising to find such an extensive displacing lesion over the left hemisphere in a right-handed individual without the slightest suggestion of aphasia.

When mental symptoms, fever, stiff neck, and weakness of the left arm became evident one week after operation, it was natural to suspect meningitis or a subdural hematoma on the right which might have been overlooked. The improvement which followed repeated drainage of blood-tinged spinal fluid suggests that cerebral edema and the irritative effects of blood in the subarachnoid space may well account for this temporary, yet surprisingly delayed, complication.

CASE 9.—Indefinite latent interval following head injury. Personality change and delirium. Fluctuating periods of lethargy and alertness. Left-sided paresis and left Babinski. Wassermann positive in the blood, negative in spinal fluid. Bilateral trephine. Chronic subdural hematoma on left. Recovery.

History: On September 7, 1935, William D., a 22-year-old colored porter, was transferred from the psychopathic division because of increasing drowsiness and bilateral papilledema.

There was a history of a primary chancre in 1929 for which he received very little treatment.

He was struck on the head during a street fight. For two days he had a mild headache, but then started to act "queerly" at home. At a nearby hospital a diagnosis of "fractured skull, and C.N.S. lues," was made. After several days he became delirious and was therefore transferred to the psychopathic division. Here his condition changed to one of fluctuating somnolence. There was very little change for ten days.

Examination: The patient was lethargic but at times could be aroused very readily. He had a rigid neck and bilateral Kernig reaction. Several scars of long standing were found over the scalp and face. The pupils were small, equal, and reacted sluggishly to light. Both fundi showed advanced papilledema with tortuous and engorged vessels. No facial asymmetry was noted. Only a mild left hemiparesis was demonstrable. The left knee jerk was more active than the right, and the abdominal responses on the left could not be elicited. Babinski sign was positive only on the left.

Laboratory data: Blood Wassermann reaction was positive. Spinal fluid was xanthochromic and showed a pressure of 500 mm. When the lumbar puncture was repeated two days later, the fluid was clear, the pressure was only 340 mm., showed only 8 cells, all lymphocytes, total protein was 50 mg. The Wassermann reaction was negative. X-ray films of the skull showed erosion of the posterior clinoids and posterior displacement of the pineal shadow.

Course: The morning following the second lumbar puncture, the patient became alert, responded promptly, and spoke rationally. By 7:00 P.M. that evening he was again in deep stupor and could not be aroused by strong painful stimulation. At 8:45 P.M. he was awake and talking. The following morning he was alert, talkative, cooperative, and facetious. By 1:00 P.M. he was again very drowsy.

Opinion: Because of the positive blood Wassermann it was thought for a time that the entire picture might be explained on the basis of meningovascular syphilis. Against this point of view was the negative spinal fluid Wassermann, and the absent pleocytosis in the spinal fluid.

Operation: Sept. 24, 1937, under local novocain anesthesia, bilateral trepanation was performed over both postparietal regions. The exposed dura on the right did not pulsate but was normal in color. When the dura was opened, the brain was seen to be under increased tension.

The exposed dura through the left trephine hole also failed to pulsate and appeared yellowish. Incision of the dura showed it to be thick and firm, and when it was retracted an encapsulated bluish mass came into view. The capsule was incised and about 60 c.c. of chocolate colored fluid was aspirated. Irrigation was continued in various directions with warm normal saline through a No. 14 catheter until the return washings were clear.

Postoperative course: His improvement started almost immediately following the removal of the hematoma. Before the first week was over, the left-sided paresis and stiffness of the neck had disappeared and the fundi showed subsiding papilledema. A week later he was out of bed and antisyphilitic therapy was started. He was discharged from the hospital four weeks after the operation, at which time he was in excellent condition.

Comment.—The positive blood Wassermann was responsible for the delay in operative intervention. This patient very strikingly portrayed fluctuating periods of consciousness, from deep stupor to alertness and back to a drowsy state, changing within several hours. Careful observations of the state of the pupils unfortunately were not recorded. Perhaps they varied only slightly because of his syphilitic condition. The paresis and the hematoma were on the same side.

CASE 10.—Injury to frontal region without loss of consciousness. Latent interval of a day, then headache, vomiting and personality change. Under observation for a week on psychopathic wards. Stupor and left-sided paresis of rather acute onset. Xanthochromic spinal fluid. Bilateral trephine. Evacuation of subdural hematoma on right. Complete recovery. Sudden death three months later. Autopsy showed no recurrent hematoma and complete absorption of subdural membrane.

History: On Nov. 11, 1935, Michael S., a 76-year-old man, was brought to the psychopathic division of the hospital because of some change in personality.

Three days before admission, while crossing the street, he was struck down by an automobile. He fell on his face and bled from the nose. He did not lose consciousness. Upon arising he felt pain over the right side of the chest, over the right shoulder, and in the left ankle. However, he managed to walk home by himself, a distance of three streets, though he felt weak and somewhat dizzy.

The next day he began to complain of headaches and pain over the lower right chest upon inspiration. For two days he vomited everything taken by mouth. Change in personality as well as some confusion, disorientation, and poor memory were first noted on the day preceding his admission to the psychopathic division of the hospital.

Examination: This 76-year-old man was able to walk into the psychopathic ward although he complained of pain in the left ankle.

General examination showed an elderly man with advanced arteriosclerosis, arcus senilis, and bilateral cataract. Blood pressure: 164, systolic; 80, diastolic. Over the aortic region there was a loud, rough, snapping systolic murmur. In addition

to ecchymosis about the right shoulder there was swelling and tenderness over the left external malleolus. Over the lower right chest anteriorly there was exquisite tenderness.

The positive neurological findings were: (1) stiffness of the neck and bilateral Kernig, (2) pupils, right larger than left, both reacting well to light and accommodation, (3) no papilledema or facial weakness, (4) absent abdominal responses, positive Babinski on right.

Fig. 7.

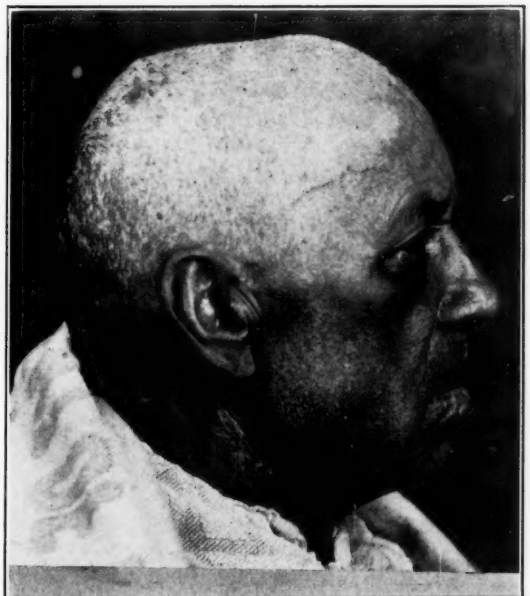

Fig. 8.

Fig. 7 (Case 10).—Healed curved scalp incision over right postparietal area.

Fig. 8 (Case 10).—Specimen three months after operation. No recurrence of hematoma. Capsule completely absorbed.

Course: Eight days after admission the patient was found in a deeply stuporous condition and incontinent. Examination at this time showed ecchymosis about the right eye, a left facial weakness, a left hemiparesis, absent abdominal responses, and positive Babinski on left.

Spinal fluid was xanthochromic, recording a pressure of 190 mm. After the removal of 5 *c.c.*, it dropped to 110 mm. Following the removal of the fluid, respirations became Cheyne-Stokes in type with period of apnea of distressing duration. He was promptly given 100 *c.c.* of 50 per cent glucose intravenously and immediately taken to the operating room.

Operation: Under local novocain anesthesia a curved transverse incision was made over the right postparietal region (Fig. 7). A trephine hole brought to view a greenish blue discoloration of the dura under marked tension. Upon opening the dura there was a fine membrane which was easily pierced with the immediate escape of brownish black fluid. The trephine hole was enlarged to about the size of a quarter; a catheter No. 14 size was introduced into the subdural sac and 60 to 70 *c.c.* of dark brownish fluid was aspirated. Irrigation with warm normal saline was continued until the return flow was perfectly clear. The brain approached the dura, and about this time the patient regained consciousness, started to speak, and began to move the left arm.

In spite of the remarkable change in the patient's condition, trepanation nevertheless was performed over the left postparietal region exposing normal appearing dura, and normal underlying brain. By the time the head dressing was applied, the patient was able to sit up and carry on a clear conversation.

Postoperative course: It was very gratifying on the second day after the operation to hear the patient's clear account of the accident. X-ray of the leg showed an oblique fracture of the left fibula in good position. On the fourth postoperative day he was allowed up in a wheel chair. By the end of the week there were no abnormal neurological signs.

The patient was discharged shortly thereafter with a cast to protect the fractured fibula.

Follow-up note: Three months after discharge, while sitting quietly in his favorite chair, he sent his daughter for some lunch. When she returned, she found him dead. We were fortunate in obtaining an autopsy, which showed advanced coronary sclerosis. The brain (Fig. 8) showed no recurrence of any bleeding. Except for slight staining of the under surface of the dura, there was no trace of any membrane or unusual vascular disturbance.

Comment.—In spite of this patient's age and critical condition there was no hesitation in performing the simple procedure of bilateral trepanation which can be done rapidly and with practically no shock to the patient. A craniotomy might well have tilted the scale against him. The post-mortem specimen offered an unusual opportunity to check upon the final result after a three-month interval.

CASE 11.—Head injury during influence of alcohol. Headache, vomiting, and lethargy. Nystagmus, diplopia, and right external rectus weakness. Advancing papilledema. Blood-tinged spinal fluid under increased pressure. Progression of symptoms under observation. Bilateral trepanation. Subdural hematoma on right evacuated. Recovery.

History: On Jan. 3, 1936, Morris O., a clerk, aged 21 years, was brought to the hospital because of severe headaches for the past two days. On New Year's morning at 3:30 A.M., after having consumed considerable liquor, he fell down some stairs. He was found in a drunken condition at the foot of the stairs with some

blood clots about the nose. The next afternoon, upon awakening, he complained of severe frontal headache, photophobia, and deafness in the left ear. On trying to walk he felt extremely dizzy. He vomited twice. X-ray of the skull suggested a possible fracture of the foramen magnum. It seemed most advisable to have him under hospital observation.

Examination: The boy appeared drowsy and uncomfortable. Over the left temporoparietal region there was an abrasion about 1 inch in diameter. There was some nuchal rigidity but no suboccipital tenderness. The pupils reacted promptly to light and convergence. The fundi showed some blurring of the disk margins. Movements of the right external rectus were limited. The patient stated he saw double upon looking to the right. There was transient horizontal and vertical nystagmus. Hearing on the left was impaired and the left eardrum was reddened.

Laboratory findings: The pulse varied from 60 to 72 per minute. Blood pressure was 145 systolic, 85 diastolic. The spinal fluid was under 320 mm. of water pressure, appeared pinkish, and showed 2,480 red blood cells per c.mm.

Course: Headaches and occasional vomiting continued. After three days the lumbar puncture was repeated; the initial pressure was 200 mm. and showed 510 crenated red blood cells.

The fundi at this time showed progression in papilledema. Withdrawal of spinal fluid or repeated infusions of intravenous glucose brought only slight relief from headache.

One week after admission the spinal fluid showed an initial pressure of 300 mm. There were no cells, but the fluid was xanthochromic. Four days later another lumbar puncture revealed a pressure of 208 mm. There were no cells and the fluid was still xanthochromic.

On Jan. 15, 1936, he complained of some numbness in the left upper extremity but this was not associated with any objective findings. The fundi showed 2 diopters papilledema. The boy appeared more lethargic and his general condition became progressively worse.

Operation: Jan. 16, 1937, trepanation over the left postparietal region disclosed normal appearing dura under tension. The underlying brain appeared injected, but there was no evidence of a hematoma.

When the dura was exposed on the right, it had a greenish-yellow discoloration. Upon opening the dura there promptly came to view a dark brownish encysted mass. This contained over 40 c.c. of dark yellowish brown fluid. In addition there was about one ounce of old clotted blood. Irrigations with warm saline were directed to all parts of the encysted cavity. At first the brain was sunken about one inch below the surface of the dura, but as more and more of the clot was removed, and the washings became clearer, the brain gradually rose to the surface and began to pulsate.

Postoperative course: There was a temporary rise in temperature to 103° on the day following the operation. The next day there was notable improvement; the patient was alert, no longer complained of headache or diplopia. Papilledema began to recede. Lumbar puncture performed five days after operation showed a pressure of 110 mm. with 260 red blood cells per c.mm. On Jan. 30, 1936, the patient was discharged, symptom free with no abnormal neurological findings, except for slight impairment in hearing on the left.

Comment.—From the time of admission, two days after the injury, to the day of operation, some thirteen days later, we had the unusual opportunity of observing the development of a chronic subdural hematoma. Headache and somnolence were the outstanding symptoms.

These steadily progressed and soon diplopia, external rectus weakness, and nystagmus appeared. The spinal fluid changed from being slightly blood tinged to a xanthochromic color, but the pressure was consistently high. To be sure, the capsule of the hematoma was very thin, but yet it was very definite, and its contents showed clotted and liquefied blood.

CASE 12.—Mild head trauma in a hypertensive individual. No loss of consciousness. Progressive headaches, dizziness, and semistupor. Ptosis on right, left hemiparesis, left facial, and left hyperreflexia. Bilateral Babinski. Spinal fluid under increased tension. Ayala index 2.5. Bilateral trephine, subdural hematoma on right evacuated. Fall in blood pressure. Recovery rapid and complete. Hypertension gradually returned.

History: Philip B., a bicycle repair man, aged 58 years, was brought to the hospital in coma. For thirteen years he was known to have had hypertension. At fairly regular intervals he consumed large quantities of liquor.

Ten days before admission, while under the moderate influence of liquor, he slipped and struck the left side of the forehead against an iron bar. There was no loss of consciousness. That night he complained of headache, but the next morning he returned to work and after a few hours the headaches became so severe that he had to go home. During the ensuing week he remained in bed complaining of headaches and dizziness. On the morning of admission he could hardly be aroused.

Examination showed a middle-aged plethoric individual with flushed face and cyanotic lips, lying quietly in a stuporous condition. Painful stimuli only partially aroused him. The neck was stiff and Kernig sign was positive on both sides. There was moderate peripheral arteriosclerosis. Blood pressure: systolic, 210; diastolic, 120. The heart was slightly enlarged to the left.

The pupils were small, irregular but equal and reacted well to light. There was a ptosis of the right upper lid. The fundi showed retinal arteriosclerosis of the hypertensive type. A few medullated nerve fibers were present on the left, but the disk margins showed no elevation. There was a left central facial weakness in association with a left hemiparesis. The knee jerks and biceps responses were more active on the left. The left ankle jerk could not be obtained. Abdominal reflexes were absent. Babinski sign was present on both sides.

Laboratory data: Except for a moderate trace of albumin, the urine was negative; blood sugar, 85 mg. per cent; urea nitrogen, 7 mg. per cent. Spinal fluid was xanthochromic with an initial pressure of 240 mm. After the removal of 10 c.c. of fluid, the pressure reading was 60 mm.; Ayala index, 2.5;* total protein, 74 mg. per cent; sugar, 65 mg. per cent; and chlorides, 60 mg. per cent.

Preoperative considerations: One had to consider whether this man with a known hypertension for thirteen years fell and struck his head as a result of a spontaneous cerebrovascular accident or whether the injury was an independent etiologic factor of a subdural hematoma. The increased spinal fluid pressure and Ayala index pointed in favor of an expanding intracranial lesion. Exploration seemed warranted.

*The Ayala index is computed by multiplying the ratio between the initial and final spinal fluid pressures by a constant quantity of fluid removed. The quantity of fluid must be 10 c.c. or more. In a recent study of 176 consecutive cases to determine the presence or absence of an expanding intracranial lesion, Savitsky and Kessler[13] found that the Ayala index was a dependable guide in 84 to 88 per cent. An Ayala index below 5 favors the diagnosis of an expanding intracranial lesion. If the index is above 5.5, it favors the diagnosis of a nonexpanding lesion. Case 8 is a good example of how the Ayala index aided in the solution of the diagnosis which was made complicated by the factor of hypertension.

$$\frac{\text{Final pressure (60 mm.)}}{\text{Initial pressure (240 mm.)}} \times \text{No. of c.c. of spinal fluid removed (10 c.c.)} = \text{Ayala index (2.5)}$$

Operation: On July 13, 1936, the afternoon of his admission, under local novocain anesthesia, bilateral trepanation was performed over both postparietal regions. The exposed dura on the left was under moderate tension, did not pulsate, and appeared normal in color. The underlying brain showed no abnormality.

When the dura was exposed through the trephine hole on the right, it appeared light yellowish green. Shining through the dura was a definite bluish discoloration. The bone defect was enlarged to the size of a quarter. When the dura was incised, there immediately came to view "current-jelly clots." A catheter was inserted into the subdural space and by aspiration 35 c.c. of dark brown fluid was obtained. Irrigations with warm saline were directed to all parts of the subdural space until the return washings were clear. While this was being done, the patient became more alert and began to speak. The wound was closed without drainage. The blood pressure in the meantime dropped to 145 systolic, 105 diastolic.

Postoperative course: The following day the patient was alert and even facetious. Motion in the left arm and left leg was improving. The left facial weakness was less distinct.

By the third postoperative day power to the left side of the body returned to almost normal. The facial weakness was only slight. The patient was alert, active, and anxious to get out of bed. Blood pressure: systolic, 174; diastolic, 112. Within the first week after operation, the blood pressure rose to systolic 180, diastolic 120, and at the time of discharge, two weeks after operation, the blood pressure was systolic 196, diastolic 110.

When seen five months after his discharge, he appeared in excellent condition. On one occasion during this interval he had a mild fainting spell at which time he noted tingling of the left hand. From this he recovered promptly. There were no abnormal neurological signs. Blood pressure: systolic, 252; diastolic, 128.

Comment.—In this patient one wondered which came first, the hypertension or the hematoma. In the end it proved that both were present and no doubt were independent.

Also, we have come to rely upon the Ayala index as an indicator of an expanding intracranial lesion. In this patient, particularly, it was put to a crucial test and proved very valuable.

CASE 13.—Headaches and personality change for a year. Severe headache, blurring vision, diplopia one month. Injury two weeks previously, rapid progress of symptoms. Dilated pupil on right, bilateral papilledema, right facial weakness, spasticity both legs, retropulsion, xanthochromic spinal fluid under increased pressure. Ventriculograms showing ventricles displaced to left. Craniotomy. Multilocular chronic subdural hematoma on right in association with meningioma. Immediate recovery excellent. Sudden death. Pulmonary embolism.

History: Sam M., a 50-year-old man, came to the hospital because he repeatedly fell backward when trying to walk forward. For about a year there was a gradual slowing up in his activity and he showed mild changes of personality and memory defects. Two months before admission he began to complain of blurring vision and diplopia. Headaches were rather severe for a month. Two weeks before entry he felt faint and fell; he had to be picked up but managed to get home unescorted. Since then he has fallen several times. When he attempted to walk forward, he fell backward. Dizziness, disorientation, weakness, and urinary retention were the most recent symptoms. There were no convulsions.

Examination showed a man who appeared much older than his age with evident congenital malformations of both hands and right foot. He was alert, cooperative, but child-like in his responses. Blood pressure: systolic, 150; diastolic, 70.

The positive neurological findings were: right pupil larger than left, bilateral papilledema, bilateral external rectus weakness, suggestive right facial weakness, spasticity of both legs, more marked on the left. Gait was with a wide base with marked retropulsion. Babinski sign was doubtful on both sides.

Laboratory data: Urine showed a slight trace of albumin but was otherwise negative. Red blood cells, 4,200,000; hemoglobin, 78 per cent; leucocytes, 7,600 per cubic mm. with 69 per cent neutrophiles and 24 per cent lymphocytes. Spinal fluid was xanthochromic under a pressure of 160 mm. of water. After the removal of 10 c.c. of fluid, pressure was 110 mm. Ayala index, 6.9; 2 lymphocytes. Pandy zero, Wassermann negative.

Ventriculography: On July 2, 1936, the patient was suspected of having a right frontal neoplasm. Trepanation was performed over the left postparietal region. The dura appeared under tension. Forty cubic centimeters of clear fluid was obtained from the left ventricle, and 30 c.c. of air was replaced. Roentgenograms of the skull showed marked displacement of both ventricles and interventricular septum to the left. The right lateral ventricle was markedly compressed, the left one was dilated.

Operation: July 6, 1936, a large right frontotemporoparietal craniotomy was performed. The exposed dura surprisingly enough did not appear to be under undue tension. Its color was natural except for a greenish tint in the region of the temporal lobe. As the dura over the right frontal region was incised, there immediately came into view a bluish cystic membrane which was adherent to the under surface of the dura. The dura was gently separated from the membrane and reflected toward the midline, exposing the hematoma which occupied the entire operative field and tapered off toward the tips of the frontal and temporal lobes. Dissection of the hematoma revealed that there were really three different stages of decomposition and organization. In all there was about 2½ ounces of old decomposed blood. Although a direct communication could not be seen between the sacs, yet they were so intimate that differences in osmotic pressures could account for the different characteristics of their contents. The membrane nearest the brain surface was not adherent and peeled off readily except at one point where a large branch of the Sylvian vein was attached to the cyst wall. This might well have been the point of recurrent venous bleeding. The surface of the brain was depressed after the removal of the hematoma, but the convolutions appeared normal. An additional finding was a small meningioma, about the size of a hazel nut, which was attached to the under surface of the dura close to the longitudinal sinus. The tumor was easily removed. At the close of the operation, the patient was given a transfusion of 500 c.c. of blood.

Postoperative course: During the first week after the operation the patient presented the mental picture of a Korsakoff psychosis. He was restless, confused, disoriented, and confabulated. The spinal fluid showed a pressure of 170 mm. of water and the fluid was slightly yellow.

The patient's condition then changed; he became mentally clear and was well oriented. He was able to give an account of his fall. His memory showed signs of improvement. Papilledema began to recede. The wound was healed by primary union and the patient was about ready to sit out of bed when on the tenth postoperative day he complained of feeling weak and promptly died.

Post-mortem examination showed a large pulmonary embolus. The operative site showed no recurrent bleeding.

Comment.—Had it not been for the presence of papilledema, the increased spinal fluid pressure, and the xanthochromia, one could

readily be satisfied in this patient with the diagnosis of chronic encephalitis. The marked ventricular displacement was not in harmony with the insignificant pyramidal tract disturbance and raised the suspicion of a chronic subdural hematoma. The finding of multilocular cysts was surprising. The direct attachment of a vein to the cyst wall has been encountered in a previous craniotomy for a subdural hematoma (Case 5). The possible causal relationship of the small parasagittal meningioma is speculative.

CASE 14.—Mild head trauma to forehead. Headaches, dizziness, unsteady gait, increasing somnolence. Lucid interval three months. Inconstant neurological signs. Encephalogram showing marked ventricular shift to the right. Removal of chronic subdural hematoma through enlarged trephine. Serial x-ray studies of absorption of air from subdural space, and shifting back of ventricles to normal position.

History: On February 15, 1935, Morris D., a 47-year-old man, entered the hospital with the following story. Three months previously he had an attack of "grippe." During this illness he had a spell of dizziness and weakness, felt nauseated, and fainted. As he fainted, he fell and struck the left temporal region against a door. There was no abrasion or period of unconsciousness. Ever since the fall he noted constant pain over the left temporal region. He had frequent periods of nausea but vomited only once. He complained of spells of generalized weakness and compression about the heart. Three weeks before admission he was carefully observed at another hospital where no abnormal findings were discovered. Shortly thereafter the headaches became more severe and radiated to the occipital region and across the right side of the head. He soon became unsteady on his feet, began to see double, and became increasingly somnolent. There was no speech disturbance and no convulsions. The patient was right handed.

Examination: The man appeared drowsy but could be aroused. He was indifferent to his surroundings. Blood pressure: systolic, 110; diastolic, 70. Pulse varied from 46 to 60 per minute. The pupils were equal in size most of the time, but on several occasions the left one was larger. The fundi were within normal limits and there was only a questionable weakness of the right side of the face. He was slightly unsteady in gait and there was ataxia in performing the right finger-to-nose test. The knee jerks on the right were more active than the ones on the left. The abdominal responses on the right could not be obtained. Babinski sign was at times positive on the right and at other times found on the left.

Laboratory data: The urine was negative. Red blood cells numbered 5,200,000 per c.mm.; hemoglobin, 90 per cent; leucocytes, 10,300 with 64 per cent neutrophiles and 25 per cent lymphocytes; blood Wassermann, negative. Spinal fluid was clear and colorless, under pressure of 100 mm. of water, showing only 3 lymphocytes per c.mm. Pandy test was zero; total protein, 30 mg. per cent; Wassermann, negative. X-ray films of the skull showed no evidence of increased pressure or other abnormality.

Encephalograms: Six days after admission, because of the fluctuating somnolence and inconstant neurological signs, encephalography was performed; 135 c.c. of clear fluid was removed and 125 c.c. of air replaced. Roentgenograms of the skull (Fig. 9) showed marked displacement of the ventricular system to the right. The left lateral ventricle was distorted and its displacement was most marked anteriorly but also partly downward. The right lateral ventricle was increased in size. The third ventricle was displaced to the right and compressed. The fourth ventricle appeared normal. The subarachnoid markings were absent on the left side of the skull but appeared normal on the right.

Fig. 9.

Fig. 10.

Fig. 11.

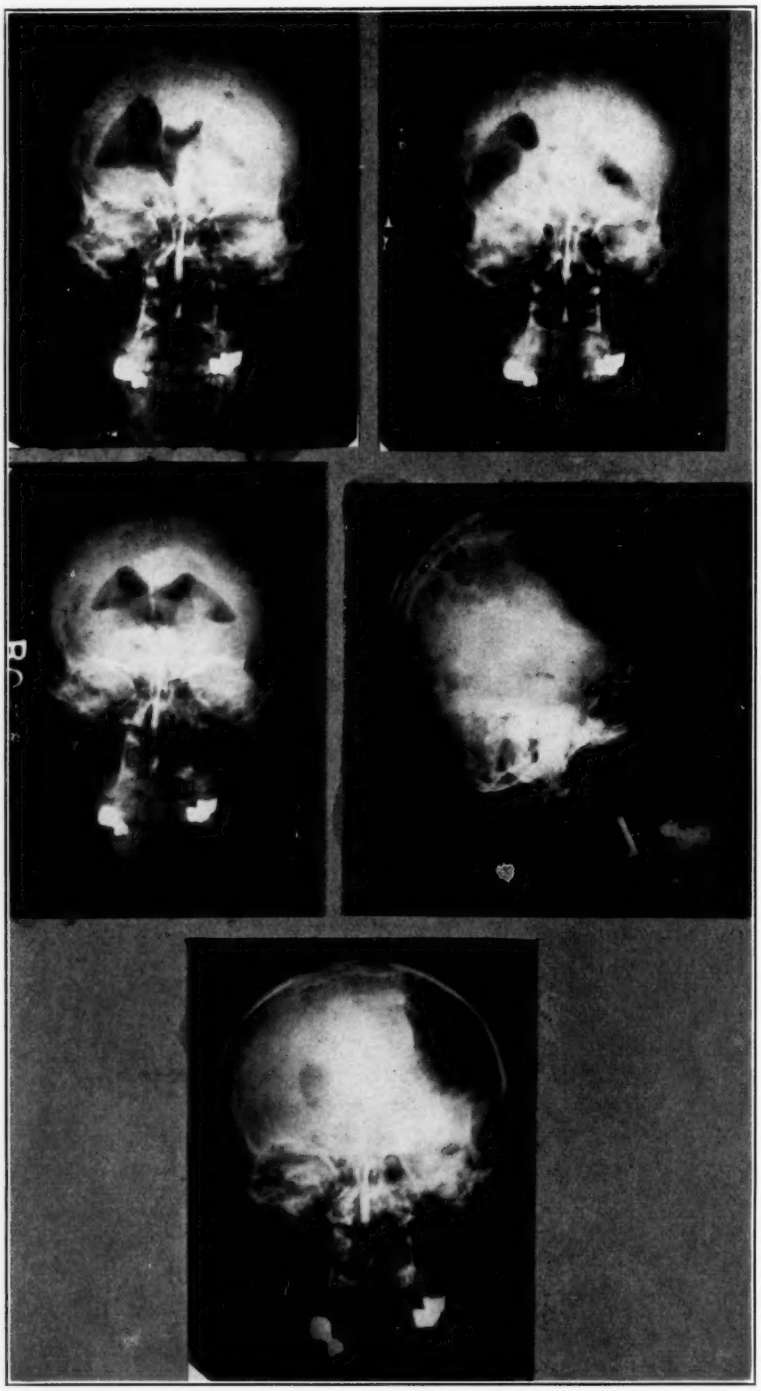

Fig. 9 (Case 14).—Encephalograms A-P and P-A views. Marked ventricular displacement to the right.

Fig. 10 (Case 14).—Immediate postoperative films showing migration of ventricles to natural position. Air in subdural space outlining the extent of the hematoma.

Fig. 11 (Case 14).—Films one week after operation. Small quantity of air in right ventricle. Absorption of air from subdural sac much slower.

The possibility of a chronic subdural hematoma was strongly considered although a neoplasm could not be definitely ruled out.

Operation: Feb. 23, 1935, a large left temporofrontoparietal flap was outlined preparatory for a craniotomy. A single trephine hole was first made through a small skin incision over the postparietal region. The exposed dura had the normal shiny luster but was under tension and did not pulsate. When the dura was incised, there came into view the typical bluish membrane of a cyst wall. The cranial defect was enlarged to about the size of a half dollar and the dura then incised in radial fashion. When the cyst wall was punctured, there was an immediate escape of about 120 c.c. of dark brown fluid. Warm saline irrigations through a catheter were then directed to the various limits of the subdural sac. These were continued until the return washings became clear. A portion of the cyst was removed for microscopic study and was subsequently reported as showing organization of a subdural hematoma. The wound was closed without drainage.

Postoperative course: There was prompt improvement in the patient's mental condition. He became more alert, and no longer complained of headache. There was no speech disturbance. Subsequent films of the skull (Fig. 10) showed air in the subdural sac and migration of the ventricles back to their normal position. Repeated x-ray studies (Fig. 11) showed that the air was gradually absorbed in twelve days. He was discharged three weeks after operation, symptom free, and with no abnormal neurological signs.

Follow-up note two years later: Shortly after his discharge he returned to his previous job and has remained active since.

Comment.—Favoring the diagnosis of a chronic subdural hematoma was the history of a mild head trauma as well as the disproportion between the extensive ventricular displacement as shown by encephalography and the few neurological signs. A patient with a neoplasm producing such marked ventricular deformity would most certainly have exhibited an aphasia and a right hemiplegia.

Films taken postoperatively clearly showed the extent of the hematoma, the degree to which the cerebral cortex was compressed, and the return of brain and ventricles to their proper relationship.

CASE 15.—No history of head trauma. Headaches, fatiguability, personality change, somnolence. Cachectic appearance, lethargic periods, bradycardia, left ptosis, right facial weakness, no pyramidal tract signs. Ventriculograms normal. Metastatic neoplasm suspected. Left frontal exploration. Chronic subdural hematoma evacuated. Recovery.

History: Abe B., a 52-year-old glazier, was admitted to the hospital with the complaints of mild generalized headaches, fatigability, and listlessness, all of one month's duration. During this period the family noted a change in personality and a slowing up in mental processes. Shortly before admission he became unsteady on his feet, dragged the right leg, and speech became thick. He became increasingly drowsy. There was no history of trauma. The patient was right handed.

Examination: The patient was in a lethargic condition but could be aroused easily. He appeared very much emaciated. The lips were cyanotic. Blood pressure was systolic 105, diastolic 70. Pulse rate was 54 per minute. His lethargic state alternated with periods of alertness. Speech was thick and unintelligible. There was a drooping of the left upper lid, but the pupils were regular, equal, and reacted

well to light stimulation. The fundi showed slight nasal blurring. A right facial weakness could not be disputed, but there were no other motor or sensory dysfunction that could be demonstrated. At times the deep reflexes on the right were more active than on the left, but this was not constant. The abdominal responses were diminished on the right. Plantar flexion was normal on both sides.

Laboratory data: The urine was negative. Red blood cells numbered 4,400,000 per c.mm.; hemoglobin, 82 per cent; leucocytes, 9,800 with 69 per cent neutrophiles and 24 per cent lymphocytes. Spinal fluid was clear, under 90 mm. of water pressure showing only 2 cells per c.mm. Wassermann test was negative. X-ray films of the chest and skull revealed no abnormality. Studies of the gastrointestinal tract failed to show any organic lesion.

Preoperative considerations: Although the few localizing signs pointed to a left frontal lobe lesion, yet the nature of the lesion was not very clear. Several diagnoses were considered: (1) a primary cerebral neoplasm, (2) a metastatic tumor, and (3) a chronic subdural hematoma.

Ventriculograms on July 23, 1936, showed both lateral ventricles well filled, normal in size, shape, and position. The third ventricle was also well visualized and not displaced.

Course: The patient continued to complain of headache, was drowsy most of the time, and showed a persistent bradycardia. Repeated examinations for a primary extracerebral malignancy failed to disclosed any lesion.

Operation: Aug. 20, 1936, under avertin and local novocain anesthesia, a left temporofrontal craniotomy was outlined. While joining two trephine holes over the left frontal region, the dura was found unusually adherent to the skull. As it tore at one point, there was a gush of 30 c.c. of old brownish fluid. The dura was then incised through a third trephine hole and a definite membrane with a collection of dark brown subdural fluid could easily be seen. Irrigations with warm saline through a small catheter were directed underneath the trephine holes to various parts of the subdural space until the return washings became clear. The underlying brain approached the surface of the dura, and the wound was closed without drainage.

Postoperative course: Following the operation the patient became more alert but for about a fortnight he continued to show mental changes and speech defects. He was euphoric, confabulated, and at times completely disoriented. These symptoms all cleared so that at the time of his discharge, Sept. 16, 1936, he was in excellent condition and showed no abnormal neurological signs.

Follow-up note: Feb. 2, 1937, the patient was mentally clear, his memory was excellent, he had returned to work as a glazier, and neurological examination was completely normal.

Comment.—About 2 per cent of patients with chronic subdural hematomas fail to give a history of any head injury, and this patient may well be included in that group.

A metastatic lesion seemed very likely because of the patient's cachectic state and the normal appearing ventriculograms, but, since a primary focus could not be established, exploration seemed justifiable. That a chronic subdural hematoma should fail to produce any abnormality of the ventricular system is very unusual. However, within the past year at the Mt. Sinai Hospital two intracranial neoplasms were successfully removed from patients whose ventricular systems were well outlined and showed no displacement or deformity.

CASE 16.—Trauma to frontal region, latent interval of six weeks, headaches, vomiting, and diplopia. Bilateral papilledema, bilateral external rectus palsy. Right facial weakness and right Babinski. Cerebellar neoplasm suspected. Bilateral trephine disclosing a chronic subdural hematoma on left. Removal by suction and irrigation. Recovery.

History: On Dec. 3, 1936, Joseph D., an 8-year-old schoolboy, was admitted to the hospital complaining of headache and double vision.

Three months before admission, while sitting on the running board of an automobile, the machine suddenly started and the boy was pitched forward. The top of his forehead struck the pavement and he was unconscious for ten minutes. The accident was considered a minor incident, for the boy had no complaints and returned to school on the following day.

He was symptom free for six weeks after the accident and then began to complain of frontal headaches and vomited occasionally. For two weeks he had pain

Fig. 12 (Case 16).—Postoperative film of skull showing enlarged left trephine through which hematoma was removed.

in back of the neck. Three days before admission he stated that objects appeared double and at this time his mother noted that the eyes were crossed.

Examination: The boy was alert, cheerful, and cooperative. There was a tendency to posturing the head toward the right. The pupils were dilated; the fundi showed bilateral papilledema with an elevation of 4 to 5 diopters. Bilateral external rectus palsy was present. Nystagmus was slight, inconstant, and more notable upon looking toward the left. A right facial weakness was readily demonstrable. There was generalized hypotonia but no motor weakness. The deep reflexes were sluggish on both sides. The Babinski sign was positive only on the right. There was not the slightest suggestion of any speech disturbance or visual field defect. The patient was right handed.

Preoperative impression: Because of this patient's age, the advanced papilledema, and the few localizing signs, a cerebellar neoplasm was suspected. For this reason

a lumbar puncture and spinal fluid studies were omitted. However, because of the history of trauma, ventriculograms were advised to aid in the differential diagnosis.

Operation: Dec. 7, 1936, under local novocain anesthesia, trepanation was performed over the right postparietal region. The exposed dura was normal in color but under tension. A ventricle needle was then inserted in the direction of the right ventricle, but two different attempts failed to yield any fluid. This was particularly disconcerting for with a cerebellar neoplasm one anticipates finding dilated lateral ventricles. A third attempt in a lateral direction encountered the right lateral ventricle, but only 4 c.c. of fluid could be obtained. It seemed quite evident that the lesion was on the left.

Novocain was then infiltrated over the left postparietal region; a trephine hole was made which exposed a bluish green dura. The bone defect was enlarged to the size of a half dollar (Fig. 12). As the dura was opened, there appeared a grayish red shaggy membrane. When this was opened, about 150 c.c. of old brownish blood was aspirated. Irrigation with warm normal saline was directed to various parts until the return washings were clear. The underlying brain could readily be seen at a depth of 3 cm. below the dura. The brain did not pulsate. Further inspection toward the occipital region disclosed another encysted hematoma which had a thin shiny membrane. When this was opened, about 100 c.c. of old blood escaped. This was not as brownish as the blood obtained upon opening the first capsule. Irrigations were continued as previously. The return washings became clear; soon the brain began to rise to the surface and cerebral pulsations became visible. By this time the child was in moderate shock. An infusion of saline and glucose followed by a transfusion of 200 c.c. of blood promptly improved his condition.

Postoperative course: The next day the right facial weakness became more marked, and in addition there was some weakness of the right arm. Speech was normal. The left external rectus palsy was definitely less. By the end of the first week there was no facial or motor weakness, the papilledema began to recede, and the Babinski sign disappeared. Spinal fluid was clear but under 250 mm. pressure.

At the time of discharge on Jan. 6, 1937, the fundi were normal but the left external palsy was still present. He was symptom free and had no other abnormal neurological sign.

At a recent visit he appeared in excellent condition, the left external palsy had completely disappeared, and he was ready to return to school.

Comment.—Although it is rare to find a chronic subdural hematoma in a child, yet it is even more surprising to find such an extensive collection of blood over the left hemisphere with so few localizing signs. It has been pointed out in a previous publication[12] that the brain accommodates itself to an unusual degree in the presence of the soft sponge-like pressure of encysted blood.

CASE 17.—Injury to forehead eight weeks previously. No loss of consciousness. Headache, weakness, vomiting, and somnolence. Neurological signs few and inconstant. Suspected of psychoneurosis. Xanthochromic spinal fluid. Coma. Bilateral trepanation, aspiration of bilateral chronic subdural hematoma. Postoperative anxiety state. Recovery.

History: On September 13, 1937, Celia K., a 39-year-old housewife, was brought to the hospital because of headaches and weakness. Eight weeks previously she

struck her forehead against an open door. She was dazed but did not lose consciousness. Since the injury she has complained of sharp, throbbing, generalized headache, particularly severe over the frontooccipital regions. Despite several weeks' vacation the headaches persisted; she slept very little and lost weight. On several occasions she vomited rather forcefully and sudden movements of the head brought on a spell of dizziness. For ten days before admission the headaches were so severe that she remained in bed. Memory, speech, and temperament were unchanged.

Examination: The patient lay in bed moaning and whining with headache. General physical examination failed to reveal any abnormality. Blood pressure: 133, systolic; 90, diastolic; pulse, 70 per minute. There was a tendency to keep the right eye closed. Fine tremors of the upper eyelid were observed, but there was no true ptosis. The pupils were equal and responded well to light and accommodation. A left central facial weakness was inconstant. Motor and sensory systems were intact. The deep reflexes were all active and equal. Hoffmann sign was present on the left; the abdominal responses were not consistent. There was no Babinski sign, but stimulation of the left plantar surface failed to produce normal flexion.

Her two children and the home were cause for repeated anxiety. She feared pregnancy, dreaded having another child, and almost completely abstained from sexual relations. With such a background, the vague symptoms and the inconstant neurological signs at first led to the suspicion of a psychoneurotic state.

However, on the day after admission she seemed lethargic, vomited twice, rather forcefully, became confused, and had difficulty in voiding.

Laboratory data: The urine, blood, and x-ray studies of the skull were completely negative. The spinal fluid was xanthochromic under 60 mm. of water pressure, showing 6,000 erythrocytes.

Course: Shortly after the spinal puncture the patient had projectile vomiting. She became more somnolent, the right pupil was notably larger than the left, and by evening she could not be aroused.

Operation: Sept. 15, 1937, under local novocain anesthesia, bilateral trepanation was performed over both postparietal regions. The exposed dura on the left had a greenish discoloration. The dura was incised and a thick brownish grey membrane came into view. When the membrane was punctured, about 1 ounce of dark brown blood escaped. Through a catheter, irrigations of warm saline were continued in all directions until the return washings became clear. The underlying brain could readily be seen pulsating freely as it approached the surface.

The exposed dura on the right had the normal glistening color and did not pulsate. The opening on this side also exposed a thick brownish membrane. Upon opening the membrane, there was an immediate escape of about 1½ ounces of dark brown blood. Irrigations with warm saline through a catheter were similarly performed on this side until the return washings were clear and the brain approached the surface. The patient regained consciousness before the close of the operation. Both wounds were then closed without drainage.

Postoperative course: The following day the patient was alert, free of headaches, and cooperated well. Within two days the tendency to keep the right eye closed completely disappeared. Spinal fluid was clear, under 20 mm. pressure showing 200 crenated red blood cells. On the third postoperative day she began to manifest signs of anxiety, fearing that another operation was necessary. Neurological examination was completely negative. She was discharged from the hospital on the twelfth postoperative day, at which time she was greatly improved. Her physician reports that, except for moderate anxiety about minor problems, she is perfectly well.

Comment.—The rather vague history and the indefinite neurological signs were strongly in favor of a functional disorder, but the xanthochromic spinal fluid very promptly aroused the suspicion of a chronic subdural hematoma. The vomiting and coma that followed shortly after admission would have made exploration mandatory, even had the spinal fluid been crystal clear. This was the first and long awaited case with a bilateral chronic subdural hematoma in the series. That another might soon follow was realized within a fortnight.

Case 18.—Trauma to occipital region, diplopia, nausea, vomiting, lethargy and coma, Cheyne-Stokes respiration. Left pupil larger than right and slight ptosis on left. Bilateral Babinski. Spinal fluid clear and colorless, moderate pressure. Bilateral trepanation, aspiration of bilateral chronic subdural hematoma. Recovery.

History: On Oct. 28, 1937, Lena R., a German housewife, aged 69 years, was brought to the hospital in a comatose condition.

Six weeks before admission she skidded on a rug which slipped from under her, fell, and struck the back of the head. There was no loss of consciousness, but she was dazed for about ten minutes and then went about her duties as usual. Intermittent frontooccipital headaches, which were usually worse in the morning, followed. A month later, on several occasions, the patient complained of diplopia. For two days there was nausea and repeated vomiting. On the morning of admission she became drowsy; by afternoon she was stuporous, and that night in a comatose state she was hurried to the hospital.

Examination: The patient was in coma; respirations at times were Cheyne-Stokes in type. Pulse was 72 per minute. Blood pressure: 130, systolic; 80, diastolic. There was no stiffness of the neck. The left pupil was larger than the right, and there was a slight ptosis of the left upper lid. The corneal responses were poor. The fundi were normal except for moderate vascular sclerosis. There were no ocular palsies, and no demonstrable facial weakness, motor or sensory disturbance. The deep reflexes of the upper extremities were active and equal. The knee and ankle jerks could not be elicited. There was no Kernig reaction. Babinski sign was positive on both sides.

Laboratory data: Urine and blood studies revealed no abnormality. The spinal fluid was clear and colorless, with an initial pressure of 150 mm. of water. Microscopically it showed 60 red blood cells per c.mm. Total protein was 29 mg. per cent. After the withdrawal of 10 c.c. of fluid, the pressure reading was 40 mm. Ayala index was 2.6.

Operation: Within two hours after admission, the patient was taken to the operating room where, under local novocain anesthesia, bilateral trepanation over the postparietal regions was performed. The exposed dura on the left did not pulsate but was bluish. Upon opening the dura, a fine membrane was pierced and there escaped 50 to 60 c.c. of dark brown bloody fluid which did not coagulate. The dura on the right was of normal color and showed no pulsations. As the dura was opened, a fine membranous capsule was pierced and there escaped about 100 c.c. of dark brown blood which had the same color and consistency as the hematoma on the left. The fluid did not coagulate. While irrigations with warm saline were directed through a catheter, the patient awoke and began to speak. The irrigations were continued on both sides until the return washings became clear and the compressed brain, which was sunken more than an inch, slowly approached the under surface of the dura. Closure was performed in double layer without drainage.

Postoperative course: The patient made an excellent recovery. The spinal fluid on the third postoperative day was xanthochromic and under 60 mm. pressure. She

was out of bed within a week and was discharged on the tenth postoperative day, symptom free, with no abnormal neurological findings.

Comment.—In a 69-year-old woman many causes of coma come to mind, but with a history of a mild trauma, in spite of the clear spinal fluid, a chronic subdural hematoma could only be excluded by trepanation. The patient was in such a critical state that only a simple swift procedure was all she could have possibly tolerated. This experience dramatically emphasizes the necessity for bilateral exploration when a chronic subdural hematoma is suspected and the ease with which an extensive hematoma can be evacuated through an ordinary trephine hole.

CASE 19.—History unreliable except for falling spells. Patient confused, lethargic, and disoriented. Dilated right pupil. Bilateral papilledema. Babinski on right. Clear spinal fluid under pressure. Bilateral trepanation. Chronic subdural hematoma on right. Recovery.

History: Poy C., a Chinese laundryman, aged 63 years, collapsed in the park and was brought to the hospital by ambulance on Oct. 15, 1937. A reliable history could not be obtained. In addition to language difficulty he persisted in answering "yes" and "no" to all questions. From friends it was learned that for three or four months he had had episodes of falling down in the street. He was incontinent and they tired of caring for him.

Examination showed an elderly Chinese man who appeared undernourished, confused, helpless, lethargic, and mentally deteriorating. There was evidence of peripheral arteriosclerosis and several scars over both tibia. The right pupil was larger than the left; both reacted promptly to light. The fundi showed advanced bilateral papilledema. There was no facial weakness. Although he was unable to stand and had a tendency to fall backwards, there was no evidence of any paresis. The right knee and right ankle jerks were more active than those on the left. The abdominal responses were feeble on both sides. Babinski sign was positive on the right, and the left plantar response was equivocal.

Laboratory data: Temperature, 98; pulse, 80; respirations, 18; blood pressure, 135 systolic and 98 diastolic. The urine and blood Wassermann were negative. Hemoglobin, 92 per cent; red cells numbered 5,080,000 per c.mm.; leucocytes, 8,750 per c.mm., of which 62 per cent were polynuclear cells and 32 per cent lymphocytes; N.P.N., 40 mg. per cent. The spinal fluid was clear, colorless, under 420 mm. water pressure, with 56 cells per c.mm. Total protein, 30 mg. per cent; Pandy test, negative; Wassermann test, negative.

Course: For two weeks after admission there were fluctuating states of lethargy and at times he showed surprising mental clarity. Another spinal puncture showed a pressure of 220 mm. and only 2 cells per c.mm. The fundi, however, showed advancing papilledema. The diagnosis of a left frontal neoplasm involving the corpus callosum was considered as most likely. Because of the story of repeated falls, a chronic subdural hematoma was also suspected.

Operation: On Nov. 3, 1937, under local novocain anesthesia, bilateral trepanation was performed over both postparietal regions. The exposed dura on the right appeared bluish and failed to pulsate. When the dura was incised, 50 c.c. of greenish brown fluid was aspirated. The bony defect was enlarged to about the size of a half dollar and irrigations with warm normal saline through a No. 14 catheter were directed to the various limits of the subdural sac. The cerebral cortex, which at first was considerably depressed, soon began to approach the dura as the repeated

washings returned clear. Exploration through a trephine hole on the left was negative. The wounds were closed without drainage.

Postoperative course: The patient's recovery was slow but steady. Repeated lumbar punctures showed xanthochromic fluid under increased pressure, but after

TABLE I

OUTSTANDING FEATURES IN 15 CASES OF CHRONIC SUBDURAL HEMATOMA

Total number of cases	15
Male	13
Female	2
Age	
(a) From 8 yr. to 76 yr.	
(b) Average, 45 yr.	
(c) Ten above 45 yr.	
Hematoma on right	6
Hematoma on left	7
Hematoma bilateral	2
Trauma	
(a) To frontal or occipital region in practially all cases	
(b) In 13 cases there was no loss of consciousness	
In 1 patient consciousness was lost for 10 minutes	
In the other, a history of trauma could not be established	
Latent interval (period from time of trauma to onset of symptoms)	
(a) Present in 9 cases	
(b) Varied from 2 days to 6 wk.	
Symptoms	
Headache	15
Somnolence	9
Vomiting	7
Mental changes	6
Diplopia	5
Dizziness	4
Findings	
Facial weakness	9
Extensor responses	10
Dilated pupil on side of hematoma	8
Homolateral paresis	3
Contralateral paresis	2
No paresis	10
Stiff neck	6
Papilledema	6
External rectus palsy	5
Bradycardia	1
Vascular hypertension	1
Positive blood Wassermann	1
Spinal fluid (Studied in 14 cases; lumbar puncture not done in patient suspected of cerebellar neoplasm)	
(a) Color	
Xanthochromic	7
Colorless	7
(b) Pressure	
Increased	9
Normal limits	5
Operation	
Bilateral trephine	13
Craniotomy	2
Hematoma capsule	
Thick membrane	9
Thin membrane	5
Indefinite membrane	1
Mortality	
Recovered	14
Died (pulmonary embolism 10 days postoperatively)	1

two weeks the spinal fluid became clear and showed no elevation in pressure. The papilledema simultaneously subsided. A mild left hemiparesis, which was first noted with improvement in the patient's mentality, soon cleared up. One month after operation he looked and behaved like an entirely different person. He spoke English fairly well and became a favorite with the other patients. At the time of discharge, Dec. 26, 1937, he was symptom free and there were no abnormal neurological signs.

Comment.—The papilledema and mental changes were in favor of an intracranial neoplasm, the location of which was problematical. The history of repeated falls suggested the possibility of a chronic subdural hematoma. The advantages of bilateral trepanation over the postparietal region are almost self-evident. Such a procedure served not only to locate and permit the evacuation of the hematoma, but, also in the event that a hematoma was not found, it would have provided the means to establish the diagnosis by ventriculography.

A summary of the outstanding features in the fifteen cases of chronic subdural hematoma is noted in Table I.

TABLE II

DIFFERENTIAL DIAGNOSIS OF ACUTE AND CHRONIC SUBDURAL HEMATOMAS

	ACUTE SUBDURAL HEMATOMA	CHRONIC SUBDURAL HEMATOMA
Type of trauma	Severe—often followed by unconsciousness	Slight—may be forgotten; direction of blow in anteroposterior diameter; rarely loss of consciousness
Fracture of skull	Frequent	Almost never
Source of bleeding	Arterial as well as venous Laceration of brain	Only venous bleeding No brain laceration
Symptomatology	Onset of symptoms almost immediate	Latent interval characteristic weeks to months
	Seldom any fluctuation of consciousness	Fluctuating periods of consciousness
Findings	Coma early; it persists and patient dies or it diminishes and recovery may follow	Coma is late manifestation
	Paresis, pyramidal tract signs on side opposite the lesion	Paresis, pyramidal tract signs, and dilate pupil often on same side with hematoma
	Convulsions not unusual	Convulsions conspicuously absent in entire series
	Aphasia present with lesions in left hemisphere in right-handed individuals	No asphasia in 9 cases of extensive hematomas over left hemisphere in right-handed persons
Spinal fluid	Uniformly bloody or pink	Xanthochromic, sometimes clear

One may well ask: Is chronic subdural hematoma just an advanced condition of an acute subdural hematoma, or is it an entirely distinct and separate clinical entity? What evidence there is to differentiate these two conditions is set forth in Table II.

TREATMENT

Under local novocain anesthesia, bilateral trepanation over both postparietal regions is advocated as the method of choice for the localization and removal of a chronic subdural hematoma. Failure to find the hematoma by this procedure occurred only once in this series of patients. In this unusual instance ventriculograms were performed through the same trephines and they showed no abnormality. Subsequent exploration over the left frontal region revealed a small flattened subdural hematoma which was readily evacuated with irrigations, a procedure which resulted in complete recovery.

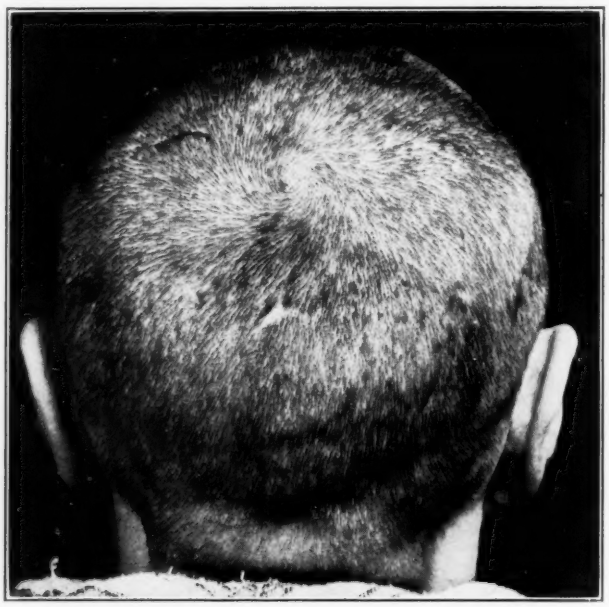

Fig. 13 (Case 18).—Operative site. Bilateral subdural hematoma.

The findings in this series of cases indicate that blood confined to the subdural space is most likely to accumulate over the postparietal region and to taper off in lens fashion to the frontal, temporal, and occipital lobes as well as toward the longitudinal sinus. As has been pointed out by Horrax and Poppen,[14] the hematoma invariably covers from two-thirds to three-fourths of the entire surface of the hemisphere. The encapsulated blood is thickest over the postparietal region, where the brain is least anchored to the under surface of the dura by traversing vessels and where it yields most readily to the spongelike pressure of the enlarging hematoma.

Incisions of the skin about two inches long are made in a curved oblique direction (Fig. 13) over both postparietal regions. Trepanation is then performed which can be used for a subsequent craniotomy should such become necessary. A greenish discoloration of the dura is a good clue to an underlying chronic subdural hematoma. At times it may be advisable to enlarge the trephine hole and extend the opening of the dura. Through a No. 14 catheter, warm normal saline irrigations are directed to the various limits of the subdural sac. The irrigations are continued until the return washings are clear. Invariably the brain will begin to rise toward the surface of the dura and then start to pulsate. Failure of the brain to behave in this manner usually means that considerable blood clot still remains. It is not necessary to remove the sac of the hematoma, unless the clot is so organized as to prevent expansion of the brain. Under such circumstances a craniotomy is most advisable. This may be done immediately or several days following the trepanation. Two patients required craniotomy following bilateral trephine explorations.

Fleming and Jones,[15] as well as Frazier,[8] have advocated four trephine holes, two on each side, with through-and-through irrigations. Although McKenzie[16] advocates the removal of the hematoma through a small bone opening by suction, he also advises drainage through a good-sized tube. Such drainage I have not found necessary.

Others, including Munro,[6] advocate bilateral trepanation over the temporal regions, which in most instances will serve very well for the localization and removal of the subdural hematoma. However, exploration through this region has several disadvantages. One is that the temporal fascia and muscle must be traversed before reaching bone, a procedure which is longer and which is often associated with considerable bleeding. Another is that trepanation over both temporal regions invariably entails separate skin preparations and redraping, which is not only time-consuming—a serious consideration in these critically ill patients—but which also involves the formidable danger of contamination. The most serious disadvantage of bitemporal exploration is realized when no hematoma is found on either side. One would then wish to perform ventriculograms, which through this region is seldom satisfactory. It is obvious that when the possibility of an intracranial neoplasm is also being considered in the differential diagnosis, a procedure which can rule out the presence of a subdural hematoma and at the same time permit the performance of ventriculograms is highly desirable.

CONCLUSION

1. A differentiation of acute and chronic subdural hematomas can and should be made.

2. Acute subdural hematomas invariably coexist with brain laceration and skull fracture. Operation should be withheld except in selective cases.

3. Chronic subdural hematoma is a separate and distinct clinical entity and not a late manifestation of an acute subdural hematoma. Operation is indicated in practically every case, and the results are excellent.

4. Bilateral trepanation over both postparietal regions is the operation of choice for the removal of the chronic subdural hematoma. With few exceptions the hematoma can be evacuated through an enlarged trephine hole. Drainage of the subdural space is not advocated. In the event of a negative exploration ventriculograms can be performed through the same trephine holes.

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THE EFFECT ON THE BLOOD FLOW OF DECREASING THE LUMEN OF A BLOOD VESSEL*

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THE idea appears to be quite general among surgeons that small decreases in the caliber of blood vessels cause corresponding reductions in the blood flow through the constricted vessels. In our work[2] with the thermostromuhr we have found that successful measurement of blood flow necessitates the application of units which have internal diameters somewhat less than the external diameters of the blood vessels under investigation. Consequently, the question has repeatedly arisen as to the probable influence of this slight constriction on blood flow through the vessel. If the slight constriction that is produced in measuring the flow of blood does not affect the flow, then the next question is: How much can one constrict a blood vessel without decreasing the flow of blood? A satisfactory answer to this question is of importance to those interested in the use of the thermostromuhr, but it should likewise prove of practical value to the surgeon to know how much a blood vessel may be constricted without decreasing the volume of blood to the region supplied.

It might be interesting to note that one can prove theoretically that, under certain assumed conditions, the radius of a blood vessel has to be reduced to approximately 0.4 mm. before the blood flow through it is reduced one-half. However, an experimental demonstration of the principle on animals is far more convincing. Consequently we are reporting here a series of experiments designed to answer the question as to the degree of constriction necessary to reduce perceptibly the blood flow through a vessel, as well as the effect on blood flow of measured amounts of constriction. In order to obtain more complete data on the problem, three series of experiments have been performed: (1) Constricting units were inserted into an artificial circulatory system. (2) Constricting units were placed in the lumen of the carotid artery of the anesthetized, heparinized dog. (3) Constricting units were applied to the outside wall of the intact carotid artery of the anesthetized dog.

ARTIFICIAL CIRCULATORY SYSTEMS

Two artificial circulatory systems were used. One was a gravity system in which the reservoir was elevated so as to maintain a pressure equivalent to the usual arterial pressure of the dog. In this system capillary tubes were used for maintaining a peripheral resistance. The

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other system made use of a constant volume pump (Dale-Schuster) and a satisfactory mechanism for maintaining peripheral resistance. In both series of experiments the constricting units were inserted directly into the system, so that the thickness of the wall of the blood vessel did not come into consideration. These constricting units were bakelite tubes, 8 mm. long, with uniform bores of different diameters. The outside diameter was approximately the size of the blood vessel. The bore varied from 0.60 mm. to 3 mm. In the gravity system the diameter could be reduced almost 50 per cent without causing more than 20 per cent decrease in blood flow when the original diameter was 3 mm. In those experiments which made use of a pump for maintaining the circulation, a decrease in diameter of 48 per cent caused a decrease in blood flow of only 4.5 per cent when the original diameter was 3 mm. Considered in terms of area of the lumen, the figures obtained in these experiments are more impressive. One can reduce the area as much as 70 per cent when using the pump system before any appreciable reduction in flow takes place (Table I). After the constriction is sufficient to cause an appreciable change in flow, additional constrictions have marked effects. In other words, the reduction in flow is rapid after a certain critical point in the degree of constriction has been reached.

TABLE I

EFFECT ON THE BLOOD FLOW OF INTRODUCING CONSTRICTING UNITS (8 MM. LONG) INTO AN ARTIFICIAL CIRCULATORY SYSTEM AND INTO THE CAROTID ARTERY OF AN ANESTHETIZED, HEPARINIZED DOG (ORIGINAL DIAMETER OF LUMEN IS 3 MM.)

PER CENT DECREASE IN:		PER CENT DECREASE IN BLOOD FLOW		
			DALE-SCHUSTER	
DIAMETER	AREA	GRAVITY SYSTEM	PUMP SYSTEM	ANIMAL SYSTEM
17	30.6	2.0	0	0
35	58.2	7.5	0	5.0
48	72.9	17.0	4.5	18.0
54	79.3	30.5	20.0	24.2
67	89.4	58.0	49.0	44.5
80	96.0	88.5	79.0	71.0

CONSTRICTING UNITS INSERTED DIRECTLY INTO LUMEN OF CAROTID ARTERY

In most of the experiments in this series the animal's blood was partially defibrinated and heparin was then added in order to make the blood noncoagulable: About 300 c.c. of blood were removed at one time. This blood was defibrinated, carefully strained through gauze, and returned to the animal intravenously. After the blood was well defibrinated, sufficient heparin was added from time to time throughout the experiment to prevent intravascular clotting. The blood pressure was recorded continuously. The devices used for constriction were the same as those employed in the first series of experiments, and they were inserted directly into the lumen of the carotid artery. This technique eliminated the necessity of estimating the thickness of the wall of the blood vessel. In these experiments it was found that a decrease in diam-

eter of about 50 per cent caused a decrease in flow of about 20 per cent when the original diameter was 3 mm. A decrease in diameter of almost 70 per cent caused, on the average, a decrease in flow of 44.5 per cent for the same original diameter. The length of the constricting units was 8 mm. The blood flow was measured directly (Table I).

CONSTRICTING UNITS APPLIED TO THE OUTSIDE WALL OF THE CAROTID ARTERY

In the experiments in this series the constricting units were 10 mm. long and were cylindrical. The blood vessel could be inserted easily through a small groove at the top of the constricting unit. The external

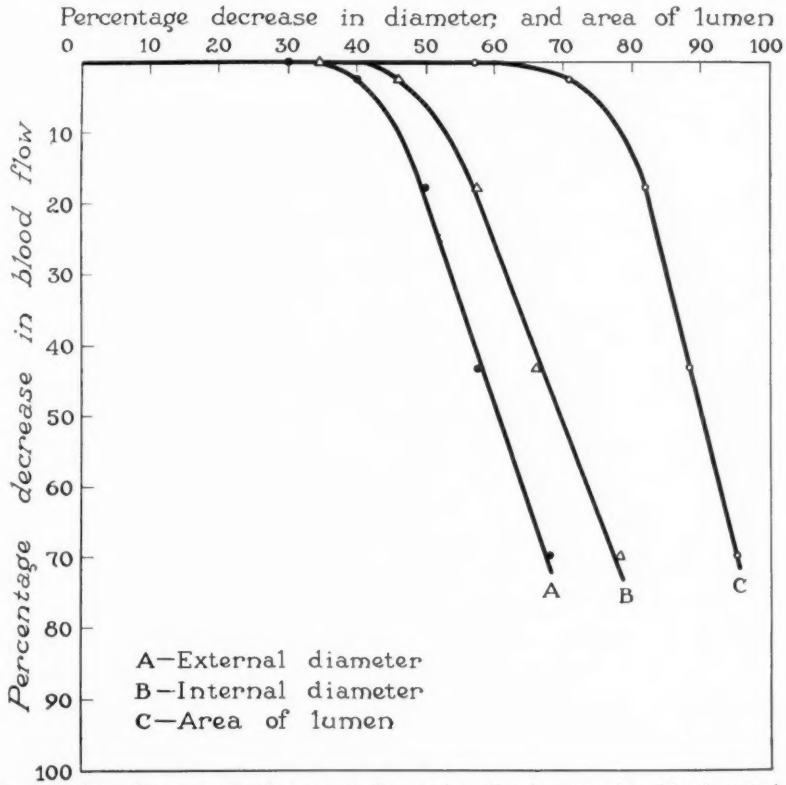

Fig. 1.—The effect on blood flow of decreasing the lumen of a blood vessel: *a*, relation between decrease in external diameter and blood flow; *b*, relation between decrease in internal diameter and blood flow; *c*, relation between decrease in area of lumen and blood flow. The external diameter of the blood vessel was 5 mm.; the double thickness of the vessel, 0.65 mm.

diameter of the blood vessel was measured by a vernier caliper. The vessel was collapsed and the thickness of both walls was measured by means of a micrometer caliper. From these measurements decreases in blood flow could be correlated with external diameter, internal diameter, and area of the lumen of the blood vessel. Four experiments were performed, all of which gave comparable results. The blood flow was measured by the thermostromuhr method. It is interesting to compare the changes in the internal diameter with those of the external diameter. The results appear much more striking if one thinks in terms of the area

of the lumen. Any change less than 10 per cent may be regarded as insignificant (Table II and Fig. 1).

TABLE II

EFFECT ON THE BLOOD FLOW OF DECREASING THE LUMEN OF A BLOOD VESSEL—EXTERNAL DIAMETER OF BLOOD VESSEL (CAROTID ARTERY), 5 MM.; DOUBLE THICKNESS OF BLOOD VESSEL WALL, 0.65 MM.

	PER CENT DECREASE IN:		
EXTERNAL DIAMETER	INTERNAL DIAMETER	AREA	BLOOD FLOW
30.0	34.5	57.1	0
40.0	46.0	70.8	2.4
50.0	57.5	81.9	17.8
57.5	66.1	88.4	43.4
68.0	78.2	95.2	70.0

COMMENT

While accurate measurement of the effect on blood flow of constriction of a blood vessel has been limited to arteries, cursory observations would indicate that the lumen of a vein may also be constricted to a considerable degree without seriously decreasing the amount of blood flowing through it. Some application of the results of this investigation can be made: In relation to our studies on blood flow, it is clearly indicated that a constricting unit can be used which has a diameter smaller than the diameter of the blood vessel. It is also obvious that the lumen at the site of an anastomosis of blood vessels can be less than the lumen of the vessel without seriously interfering with the transport of blood. Finally, it should be noted that success in the repair of injured blood vessels can be anticipated even if it is not possible to restore the lumen completely.

SUMMARY

The carotid artery of a dog may be constricted to a considerable degree before the blood flow is reduced significantly. The results may be given in terms of the reduction in external diameter, internal diameter, or area of lumen. The external diameter of a blood vessel (5 mm. in diameter and with a wall 0.325 mm. thick) may be reduced 40 per cent without a significant reduction in blood flow, but, if the external diameter is reduced 60 per cent, the blood flow is reduced 49 per cent. On the other hand, the internal diameter can be reduced 70 per cent before a 50 per cent reduction in blood flow takes place. The data are most striking when stated in terms of the area of the lumen. The area of the lumen may be reduced 50 per cent without any change in blood flow, and it can be reduced as much as 90 per cent before a 50 per cent reduction in blood flow occurs.

The data were obtained with constricting units of not more than 1 cm. in length.

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REPORT OF 500 BLOOD TRANSFUSIONS

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THE early interest in blood transfusions was increased in the seventeenth century following the works of William Harvey. So many severe and fatal reactions followed blood transfusions during this time that the procedure became very unpopular.

In the latter part of the nineteenth century small series of blood transfusions were reported; many reactions occurred, the mortality rate was high. Even though Blundell had shown that small amounts of air introduced into the circulatory system did not produce serious effects, injected air was credited as the cause of reactions. The last decade of this century saw the development of blood vessel surgery and, as a result, transfusions became more common.

Guthrie and Huck in their article "Existence of More Than Four Isoagglutinin Groups" give credit to Samuel G. Shattock, 1899, for the first knowledge of agglutination, which he believed occurred in diseased individuals. Landsteiner, in 1900, is usually given credit for the discovery of isoagglutination, and a year later showed that it was independent of health and disease and followed certain definite laws. His work revealed three blood agglutination groups.

In 1902 V. Decastello and Sturli showed that 4 of 155 patients examined did not come in the Landsteiner grouping. Jansky, in 1907, independently verified this work and stated there were four groups. Two years later Moss, independently, found human blood possessed four isoagglutinin groups.

This knowledge advanced blood transfusion therapy and decreased many of the severe untoward reactions. Severe reactions did occur and many mild reactions. Cross-matching the cells of the donor with those of the recipient was added to the routine grouping, with marked beneficial results.

Various investigators (Landsteiner, Guthrie, and Culbertson) believe that there are other than the four isoagglutinin groups or intragroup isoagglutinins. Twenty-seven combinations or groupings, according to Guthrie and Huck, are biologically possible. C. G. Culbertson advises the use of a sensitive centrifuge test in addition to grouping and cross-matching. He found two patients of the intragroup classification who could only be transfused with blood of the same intragroup type. Before adopting this procedure, a patient receiving blood from a supposedly compatible donor (tested by typing and cross-matching) had a severe

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reaction, chill, temperature of 105,° hemoglobinuria, and oliguria. Accurate blood typing and cross-matching are, without a doubt, essential before a transfusion is done.

Reactions following transfusions fall into four main classifications.

I. Severe reactions coming on during or shortly after a transfusion, giving the picture of shock associated with pain in the chest and back, chills, fever, hemoglobinuria, oliguria or anuria, are directly related with incompatibility of donor and recipient blood. The mortality in this group is high. Blood typing and cross-matching of the donor's and recipient's blood have diminished this group to a rarity in well-organized hospitals.

II. Chills and fever may occur within the first twelve hours after transfusion. The mortality is low, but the addition of chills and fever to a sick individual is of negative value. Twenty-four to forty-eight hours after the reaction, the temperature is usually similar to that prior to the transfusion.

III. A temporary rise in temperature of 2 to 3° F. is not uncommon. It returns to its former level in twelve to forty-eight hours following the transfusion.

IV. Urticaria may or may not be associated with fever. It usually is very mild but may be disagreeable for days.

The causes of these reactions concern us all as well as the investigators of the past. A misstep in one direction or another may lead to a disaster. Further investigation and care in giving transfusions should result in fewer reactions.

Causes for reactions as listed by various authors and as observed come under two main headings: (a) technical causes; (b) reactions due to the administered blood.

Technical perfection, adequate equipment, fresh sera, and accurate clerical ability are essential in selecting the right donor and seeing to it that that donor gives the blood to the respective patient.

Lewisohn believes that in his hospital service they reduced transfusion reactions from 12 per cent to 1 per cent by the centralization and efficient attention to the preparation of instruments, apparatus, and solutions used in transfusions. Attention to the preparation of solutions and instruments to be used for intravenous therapy is of prime importance. Emboli and foreign protein or toxic reactions may result from poorly prepared apparatus and solutions. The apparatus must be efficient, the needles sharp and as atraumatic as possible.

The method of administration may be a factor. Individual surgeons not familiar with a technically proper procedure of transfusing are more apt to have reactions than a well-organized team. Too slow withdrawal of blood from the donor may result in clotting; too rapid administration of blood can tax the recipient's vascular system. The administration of "preserved blood," as evidenced by the recent abundant literature, has

greatly increased severe as well as mild reactions. The temperature of the administered fluids must be considered as a cause of reactions.

Two- and three-way syringes used to connect recipient and donor are convenient at times but dangerous. The recipient's blood may be given to the donor. Severe reactions have followed such accidents. A complex apparatus is frequently associated with technical difficulties and hazards.

Incompatibility of blood grouping causes the most severe reactions due to administered blood. It has been shown that there are incompatibilities within the same group. The blood from a donor used twice or more may cause reactions; anaphylactic reactions are reported. The more times a patient is transfused, the more difficult it may become to find a donor, and reactions are more likely to occur.

Toxins contained in administered blood may have been liberated from small clots or traumatized tissue of the donor or recipient. It is felt that toxins are liberated in "preserved" citrated blood and the longer it is preserved the greater the amount of toxins there may be present. Psychic reactions of the donor may give rise to toxins.

The various blood constituents have all, more or less, been attacked as the causes for reactions.

The disease of the recipient has been felt to be the cause of reactions. Some authors do not feel that this is true. It does seem that septic cases are more liable to reactions than others. Moss stated: "No constant differences were found between the agglutinating or hemolysing abilities of sera in health and in disease."

This institution* requires of a professional donor a negative history of malaria, negative Wassermann and Kahn reactions, a compatibility according to the Moss classification, and cross-matching by means of the microscopic slide method. The slide is kept in a moist Petri dish at room temperature for fifteen minutes before examination. These same requirements are made of a nonprofessional donor, except that in an emergency a Wassermann is not done; the Kahn test is always completed. Occasionally a universal donor is used. Recipients are typed and cross-matched before each transfusion when additional transfusions are necessary.

In this group of 500 transfusions, 481 were given by the multiple syringe method, 18 by the citrate method, and 1 by the Scannell method.

Following a transfusion, the apparatus is washed well with cold water, then in soap and water (Ivory soap). It is then rinsed in tap water, dried, wrapped, and sterilized. Immediately before use, the prepared apparatus is rinsed in sterile normal salt solution, then in a sterile 2.5 per cent citrate solution and given to the operator. When transfusing "clean" cases, three 100 c.c. syringes are used; those used twice are rinsed in sterile filtered tap water, rinsed in sterile normal salt solution and in sterile 2.5 per cent citrate solution before the operator receives

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them the second time. In case an infectious case is being transfused, no syringe is used more than once and the donor is guarded from contamination.

The transfusions are usually given by the resident surgeon, aided by three interns. Occasionally an attending surgeon is in charge. A few transfusions have been done in the patients' rooms when the risk of moving the patient was of importance; otherwise they were transfused in an operating room.

The indications for the 500 transfusions are listed in Table I.

TABLE I

		PRE- AND POSTOPERATIVE	
1. Secondary anemia	342	104	79
2. Directly after prolonged major operations	66		66
3. Shock	36		34
4. Blood dyscrasias	30		
5. Jaundice	26	12	14
	500	116	193

Table II records the causes of the secondary anemias and their frequency. The transfusions administered to anemic patients due to acute and chronic infections and acute and chronic gastrointestinal hemorrhages are listed.

TABLE II

		ACUTE	CHRONIC
1. Bladder and prostate pathology*	95		
2. Pelvic pathology (female)*	65		
3. Infections	61	38	23
4. Gastrointestinal hemorrhage	24	21	3
5. Kidney disease (surgical)	23		
6. Various causes (requiring surgery)	23		
7. Pre- and post partum	15		
8. Malignancy	15		
9. Nephritis	6		
10. Trauma	5		
11. Burns	5		
12. Nutritional	5		
Total	342		

*Benign and malignant.

In Table III the major surgical conditions which were followed directly by a blood transfusion are listed. Transfusions during or following prolonged or shocking major operations are of definite benefit.

TABLE III

	NUMBER OF CASES
1. Bowel resections	31
2. Stomach surgery	15
3. Neurosurgery	10
4. Kidney surgery	6
5. Gall-bladder surgery	2
6. Amputations	2
Total	66

Table IV records the two causes of shock found in this series, the number of transfusions given, and their results.

TABLE IV

		DEFINITE BENEFIT	TEMPORARY BENEFIT	NO BENEFIT
1. Postoperative shock	34	26	6	2
2. Post-blood transfusion shock (1 case)	2	2		
	36	28	6	2

The number of transfusions given patients with blood dyscrasias is listed in Table V.

TABLE V

1. Agranulocytosis	8
2. Myeloid leucemia	8
3. Pernicious anemia	4
4. Chronic aplastic anemia	4
5. Thrombocytopenic purpura	4
6. Hemolytic familial jaundice	2
Total	30

Jaundice is an additional risk to any patient undergoing a surgical procedure and blood transfusions prior and following operations are of benefit. Table VI records the causes of the jaundice and the number of transfusions given.

TABLE VI

	PRE- AND POSTOPERATIVE		TOTAL
1. Common duct stone	8	7	15
2. Chronic pancreatitis	1	4	5
3. Malignancy with obstruction to bile passages	3	3	6
Total	12	14	26

Table VII presents the number of untoward reactions in its classification.

TABLE VII

1. Chills and fever	24
2. Fever only	22
3. Urticaria with or without fever	11
4. Shock, hemoglobinuria, or oliguria	1
	58

There were two reactions in this group of 58 reactions which gave definite evidence as to their causes. A brief account of each follows.

The cause of the severe reaction (Table VII, *4*) was easily traced to an incompatible donor. The patient had been properly grouped and cross-

matched with a compatible nonprofessional donor. A laboratory sheet with a list of the compatible and incompatible donors was placed in the patient's chart. The intern, by mistake, called the incompatible donor. The patient went into shock following the administration of 360 c.c. whole blood given by the multiple syringe method. Adrenalin and papaverine were given. The temperature rose to 105.6° F.; hemoglobinuria and oliguria developed. The next day, 500 c.c. of compatible blood were given with no harmful reaction. Nausea and vomiting prevented the oral administration of fluids; fluids were administered by other routes. A third transfusion of 500 c.c. of blood was administered nine days after the first; there was no harmful reaction. The patient recovered.

Within one-half hour of an unsuccessful attempt to give a transfusion by the Scannell method, a patient experienced chills and fever. Considerable time was consumed in giving 40 c.c. of blood. Difficulty was experienced in the venepuncture of the donor and withdrawal of blood. The donor was later used and no reaction followed. The cause or causes for this reaction may be any or all of those listed under technical causes except that which has to do with compatibility.

There is no definite knowledge as to the causes of 56 unfavorable reactions.

The 500 blood transfusions were given to 288 patients. There were 115 patients given more than one transfusion; 30 of the 58 reactions occurred in this group. Ten per cent more reactions occurred to the 115 patients receiving multiple transfusions than to the 173 who received only one transfusion. There were, however, 327 transfusions given the 115 patients, or 9.14 per cent had reactions. The group receiving one transfusion had 28 reactions, or 16.18 per cent reactions. Why such a difference in these two groups exists is not known. Accurate retyping and cross-matching before each additional transfusion no doubt prevent many reactions.

Twenty-one patients had one reaction following one of a number of transfusions. Table VIII lists the 21 cases as to the occurrence of their reactions, following the first, any between the first and last, and following the last transfusion.

TABLE VIII

	AFTER FIRST TRANSFUSION	AFTER ANY OTHER TRANSFUSION	AFTER LAST TRANSFUSION
Patients given multiple transfusions with one reaction	8	8	5

Four patients having received more than one transfusion had, respectively, more than one reaction. Table IX records the reactions in relation to each transfusion.

TABLE IX

PATIENT	NUMBER OF TRANSFUSIONS GIVEN	TYPE	METHOD	TYPE OF REACTION FOLLOWING 1ST TRANSFUSION	2ND TRANSFUSION	3RD TRANSFUSION
No. 16	2	II	Citrate Multiple syringe	Chills and fever	Urticaria	None
No. 29	3	IV	Multiple syringe	Urticaria	None	Temperature, 103°
No. 100	3	III	Multiple syringe	Temperature, 101.4°	Chill, 100.4°	Temperature, 100.8°
No. 477	3	IV	Multiple syringe	Urticaria	Urticaria	None

Nine patients received blood from universal donors; 5 were type I, 1 was type II, 3 were type III. Three patients received multiple transfusions from universal donors; 3 received blood from universal donors and donors from their own blood groups; 3 patients received one transfusion, it being from a universal donor. One reaction occurred in this group; it followed the first of four blood transfusions, the patient was a type I.

CONCLUSIONS

The purpose and method of giving 500 blood transfusions to 288 patients are reviewed.

There were 58 untoward reactions encountered; 1 caused by incompatible blood, 1 by technical difficulties, 56 without definite known causes. There was no death due to a blood transfusion.

The severe reactions, as previously described, reawakened the realization that records as to the compatibility of donor and patient must be fool-proof and as simple as possible.

The other 57 reactions indicate that there is much to be done in perfecting our technique of administration and preparation of instruments and solutions before our percentage of reactions (11.6 per cent) is diminished.

There were fewer reactions for each transfusion among the patients receiving multiple transfusions than those receiving one transfusion. The reason for this is not known, but it is felt that the retyping and the rematching of the recipient with donors before each additional transfusion is done is important in lowering the number of reactions in patients receiving more than one transfusion.

The number of patients receiving blood from universal donors in this group of 500 cases is small, but it did not indicate more frequent or severe reactions.

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BLOOD TRANSFUSION AND THE STORAGE OF BLOOD FOR EMERGENCY PROCEDURES

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IT IS authentically reported that in 1667 Jean Denys and Emmerez performed blood transfusion; the recipient was a young man and the donor, a sheep from which ½ pint (250 c.c.) of blood was taken from the carotid artery. The previous epochal work of Harvey on the circulation of blood, in 1616, and the experiments of Lower, in 1665, dealing with transfusion of blood of dogs made possible the first transfusion in which man was the recipient. Blood transfusion has been practiced periodically since 1667 and has enjoyed either favor or disfavor among physicians, depending on the success or failure of the procedure. It was in 1900 that Landsteiner showed that the reason for dangerous or fatal reactions from transfusion were attributable to incompatibilities between serum and cells of donors and recipients respectively. He proved that it is possible for the serum of one normal human being to agglutinate or hemolyze the blood cells of another individual and, further, that a donor's serum may agglutinate the recipient's cells, but the recipient's serum must not agglutinate the donor's cells.

In 1907 Jansky proved that there are four blood groups applicable to human beings and this work was later confirmed by Moss and Ottenberg. The so-called Landsteiner Groups O, A, B, and AB are also known as Groups 4, 2, 3, and 1 respectively according to the Moss classification. The latter grouping is probably the most widely used today, but it is not uncommon to utilize both systems for purposes of accuracy and to list the groups as follows: 1 (AB), 2 (A), 3 (B), and 4 (O). With the latter classification one has a double check on the group of the prospective donor's or recipient's blood, and it should not be possible to confuse the Moss and Jansky systems of nomenclature. Group 4 (O) is the type of blood obtainable from that person who is known as the "universal donor," and Group 1 (AB) is the type of blood which is compatible with all the other groups, and is therefore recognized as blood of the "universal recipient."

There is considerable variation of opinion regarding the practice of using a universal donor, Group 4 (O), for any and all transfusions. It is held by some workers that Group 4 (O) blood should be used only in case of extreme emergency or when recipients of Group 4 (O) require transfusion, and that whenever possible blood of recipients and donors should be of the same group. This opinion is probably one of the

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soundest, because the supply of universal donors, Group 4 (O), is easily depleted if they are used routinely. Furthermore, the incidence of untoward reactions will be decreased in a large series of transfusions if recipients are given blood of homologous groups. Other factors, such as the method of transfusion, whether direct or indirect, and the agent used to prevent coagulation, likewise will influence the number of reactions to transfusion.

On account of the flexibility of the indirect (citrate or other anticoagulant) method of transfusion, it is usually preferred to the direct method. The relative incidence of reactions when the direct method is used is slightly less than when indirect methods are used, but the practicability of the direct method is not sufficient to warrant its routine use when a large number of transfusions are performed. Furthermore, many recipients object to the intimacy of direct transfusion and will not submit to that method. Likewise, now that the storing of blood is rapidly becoming an important feature in transfusion, the direct method is less attractive than it was.

STORING OF FRESH BLOOD

The work done on blood of cadavers[17, 20, 21] is the basis on which storage of fresh blood has been developed. It has been shown experimentally that when blood of cadavers, kept at 17 to 20° C., is substituted for as much as 60 per cent to 90 per cent of the total blood of an animal, no serious reactions occur. Stored blood of cadavers does not appear to be of increased toxicity, provided the blood is obtained prior to ten hours after death and provided it is kept cool. For transfusion, the vitality of blood of cadavers, if treated in this way, is unchanged, as is evidenced by the fact that the leucocytes within it have phagocytic power, the hemoglobin and number of erythrocytes of the blood of the recipient are increased, the serum of the blood of the cadavers behaves in the same manner as blood obtained from living persons in respect to fixation of complement, and the blood of cadavers can participate actively in the exchange of gases, one of the important and main functions of blood. Although blood of cadavers has not been used at the Mayo Clinic, such blood, obtained within ten hours after death, has been successfully used in Europe for transfusion when stored for as long as three weeks at a temperature of 17 to 20° C. There are differences, however, in the behavior of blood of cadavers and fresh blood taken from living persons in respect to coagulation; namely, blood from living persons will clot unless it is mixed with an anticoagulant; whereas, blood of cadavers, if the individuals have died suddenly from gunshot, acute trauma, apoplexy, coronary disease, or electric shock, will form a coagulum at first, but, after fifteen minutes to an hour, the coagulum dissolves and the blood becomes liquid once more. This phenomenon of

fibrinolysis can be accelerated by heating or shaking, but the mechanism is not entirely understood, since blood collected from individuals who have expired from such wasting diseases as tuberculosis, carcinoma, and so forth, does not exhibit this phenomenon and when this blood coagulates it does not again become liquid.

When the physical and chemical properties of blood of cadavers are evaluated, it is easier to understand why fresh blood obtained from living persons, in the presence of an anticoagulant, can be stored satisfactorily for purposes of transfusion.

An icebox or refrigerator of reasonable size, of which the temperature can be controlled so as to prevent freezing, is satisfactory for storing of blood. The optimal temperature is about 40° F. Refrigerated blood, three weeks old, has been administered to patients without untoward results, but the optimal time for the utilization of stored blood is two weeks or less after the blood has been put in storage. If a bottle of stored blood gives any evidence (grossly) of hemolysis, it should be discarded.

SELECTION OF DONORS

Blood of all professional donors should be carefully grouped. Also, it should be tested by at least one of the accepted procedures to detect positive or negative evidence of syphilis; in many hospitals and clinics it is customary to perform several flocculation or precipitin tests for this purpose. Tests for syphilis should be made at least every six months. Some of the most commonly used tests are the following: the Kolmer modification of the Wassermann test and the Kahn, Kline, and Hinton tests. In addition, every six months to a year, the donors should be subjected to a general physical examination which should include leucocyte and erythrocyte counts, determination of hemoglobin, and urinalysis. Donors whose veins are of large or medium size are preferred and in emergencies only those donors who have excellent veins are called. Professional donors may be used every four to six weeks, if needed, but every six to eight weeks is to be preferred. Each time the donor is called for transfusion he should be required to present a card on which are listed such data as the result of the Wassermann test, the donor's blood group, a certification of physical fitness, and the donor's signature. Some record also must be available to show when a certain donor was last used.

Nonprofessional donors may be friends or relatives of patients or good Samaritans who wish to donate their own blood. In order to determine the suitability for transfusion of blood of individuals of this class, the blood may be grouped according to the Moss or Jansky classification or, if necessary, the short method of cross agglutination may be employed. Serologic tests should be made whenever possible.

BLOOD DONOR AGENCIES

Two general methods can be employed in order to supply blood to patients who are in need of transfusion.[6, 19] Either fresh blood or stored blood can be used. When fresh blood is requested, a donor can be called through some central agency, which will direct him to the hospital or other place where the transfusion is to be performed. Many hospitals have solved this problem of obtaining blood for transfusion by establishing their own lists of suitable donors. The physicians' exchange in most cities would be an excellent bureau to assist in calling and directing suitable donors to the place of operation. A minor point, but still an important one, is that all professional donors should have telephones in their homes so that delays will be minimal. All registered donors should be supervised closely and the organization responsible for obtaining donors should maintain rigid standards in regard to periodic physical examination.

It is equally advantageous for hospitals, especially, to organize emergency laboratories so that blood from both professional and nonprofessional donors may be available for immediate use. Having stored blood on hand will expedite tremendously the average transfusion. It is not absolutely necessary to have on hand blood of all types, but there should be a reasonable supply, 1 to 2 liters, of Group 4 (O) blood, so that the patient who is in need of a transfusion can receive blood without waiting for a donor of his own group to be found or for cross matching to be performed. By the law of averages, blood of either Group 4 (O) or 2 (A) will be most prevalent and blood of either of these two groups will be available most of the time. Prior to administration of stored blood, the bottle, with its contents, should be warmed to body temperature by means of a water bath.

The problem of remuneration for professional donors is legion, and no standard regulations are possible. By and large, the average cost of blood will vary between $5.00 and $10.00 per 100 c.c. The average amount of blood taken from one donor at a time is 500 c.c. to 750 c.c. If more than this amount is required, two donors, or more, should be called.

TECHNIQUE OF WITHDRAWING BLOOD (PHLEBOTOMY)

Before withdrawing any blood from the donor, both arms should be examined to determine which vein is most suitable for phlebotomy. As was mentioned previously, only those people whose veins are of medium to large size should be used as professional donors.

After the vein to be used has been selected, the skin over the site of puncture is surgically prepared and anesthetized. In most instances the veins about the antecubital fossa are best adapted for phlebotomy. With 1 per cent solution of procaine, a wheal is raised on the skin over the vein and a tourniquet is applied above the site of puncture. A needle

of large caliber, the size of which depends on the size of the vein, is attached to a rubber tube, 15 inches (38 cm.) long and of relatively small lumen, and the needle is inserted into the vein. A 13 gauge Lewisohn needle is recommended for the larger veins, and a 15 gauge needle for veins of medium size[9, 11] (Fig. 1). Too much emphasis cannot be laid on the importance of using phlebotomy needles of adequate size and rubber tubing of which the lumen is of proper size, because most difficulties which arise in withdrawal of blood are owing to the fact that small needles are introduced into the vein and the inside diameter of the tubing which leads to the bottle that is to hold the blood is either too large or too small; under such circumstances the blood frequently clots. The tubing should be made of pure para rubber and the inside diameter of the tubing should approximate the diameter of the hub of the needle as closely as possible. Except very occasionally,

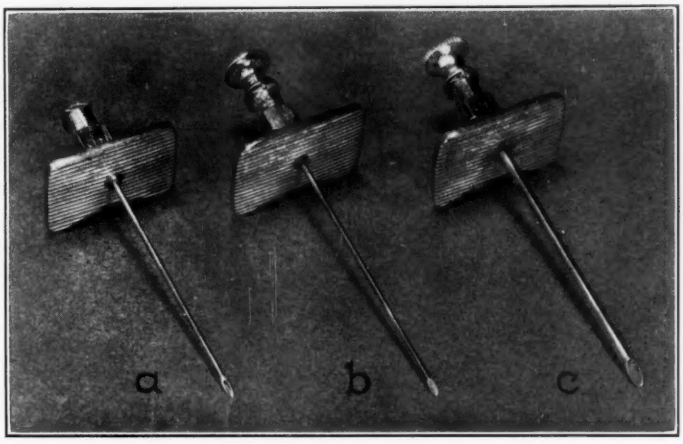

Fig. 1.—*a*, eighteen gauge Lewisohn needle with stylet removed; *b*, 18 gauge Lewisohn needle with stylet in place; *c*, 13 gauge Lewisohn needle with stylet in place.

for instance as in phlebotomy for polycythemia vera, it should not be necessary to use suction appliances to hasten the flow of blood. The donor should alternately open and close his hand while blood is being withdrawn to hasten the flow.

It is purely a matter of preference whether the point of the needle is introduced into the vein with or against the flow of venous blood.[10] Either method is satisfactory and useful.

The blood is collected in a graduated glass bottle (Fig. 2) of a capacity of 500 c.c., into which has been poured 50 c.c. of physiologic saline solution containing 18 gr. (1.16 gm.) of sodium citrate. For convenience, ampules are obtainable which contain 1.16 gm. of sodium citrate in a solution made up to 5 c.c. As an added precaution against clotting when withdrawing blood from polycythemic patients, two ampules of sodium citrate (36 gr. or 2.33 gm.) are added to 50 c.c. of physiologic saline solution.

The free end of the rubber tubing is held in one hand and the bottle is gently oscillated to insure thorough mixing of the anticoagulant with the blood. Stirring rods are not used. The bottles in which the blood is collected are capped with rubber stoppers and metal collars are screwed securely on the necks of the bottles, holding the rubber stoppers in place. There are two holes in each rubber stopper, one to allow a glass "breather" tube to extend three-quarters of the way toward the bottom of the bottle, and the other to permit a glass vacodrip adapter to be inserted when blood is administered; this latter device makes it possible to utilize the vacuum principle on which the bottle* is designed (Fig. 3).

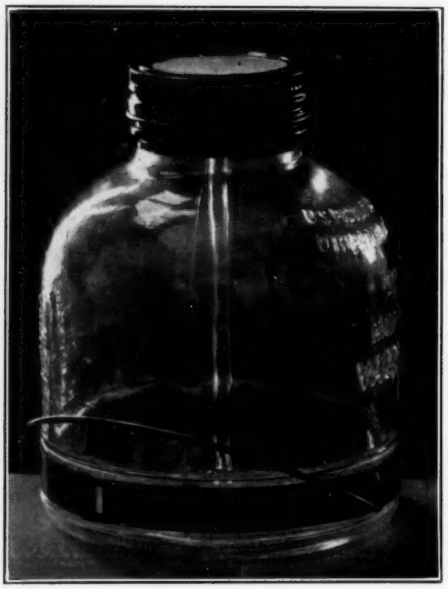

Fig. 2.—Half-liter bottle for the collecting, storing, and dispensing of blood.

ADMINISTRATION OF BLOOD

When the blood is administered to the recipient, utilization of the same bottle in which the blood was collected saves considerable time and material. The recipient's arm is prepared in the same manner as the donor's was prepared, provided the veins in the arm are suitable for venipuncture; otherwise, other veins are utilized. A needle of small bore, usually 18 or 20 gauge, is inserted into the vein and, when it is certain that the vein has been entered satisfactorily, the rubber tubing from the vacodrip bottle is connected to the hub of the intravenous needle. A maximal rate for administration of blood is 15 c.c. per minute.

In the course of many serious operations it may be found advantageous to insert into a vein a needle fitted with a stylet (Fig. 1), and to anchor the needle securely with adhesive plaster. This maneuver will facilitate

*Bottles adapted from a commercial product, Vacoliter.

intravenous therapy and will obviate the difficulties of venipuncture in the event of sudden circulatory collapse. If necessary, the stylet is removed, the needle and vein are irrigated with a small quantity of physiologic saline solution, and then blood, solution of acacia, or any other desired solution is administered. It is worth while to remember that solution of acacia should not be administered either before or after administration of blood, without first irrigating the tubing, since the acacia may cause clotting of the blood in the tubing or needle, or even in the vein.

Fig. 3.—Half-liter bottle inverted, vacodrip adapter, and rubber tubing, with adapter to fit needle.

REACTIONS TO INDIRECT TRANSFUSION

Several factors influence the incidence and number of reactions to transfusion, among which are the following: the rate of administration, the anticoagulant used,[7] the temperature of the blood, the presence of allergic antigens, the quantity of blood administered, and so forth.

The most common signs and symptoms of reaction to transfusion are: rise in temperature, chill, and urticaria. Reactions such as dyspnea and cyanosis are less common. It has been erroneously assumed that a reaction to a transfusion cannot occur if a patient is anesthetized; I wish to emphasize that reaction may occur even if a patient is under anesthesia. The reactions may not be so obvious as when the patient is not anesthetized, but the symptoms can be detected if the observer is vigilant. It is also a false impression that tetanus antitoxin can be given with impunity when a patient is anesthetized. Reactions may occur also in these instances.

Many agents have been recommended as anticoagulants; such are sodium citrate, sodium oxalate, saline solution, peptone, and heparin. Many other agents have been tried, but sodium citrate is still the anticoagulant most commonly used in the indirect method of transfusion. Considerable interesting work is being done now on the use of heparin as an anticoagulant. Recently it has been found possible to obtain larger quantities of heparin from lungs of beef than from hepatic tissue and this discovery has been a great impetus to further research with this substance.

In certain localities donors have been heparinized a day or so prior to the time when their blood was to be used for transfusion. This procedure entails injecting solution of heparin into the donor's vein for a certain period and subsequently withdrawing his blood before the heparin has lost its power as an anticoagulant or has become inactivated. It is felt that heparin, the action of which is supposedly more in consonance with physiologic processes than most other anticoagulants, will cause fewer reactions. The action of heparin when injected intravenously is to render the blood incoagulable by preventing the conversion of prothrombin into thrombin. Heparin also may be added to blood, in the same manner as is sodium citrate, but the duration of its anticoagulant action is usually limited to twenty-four hours. As yet there are not sufficient data concerning the incidence of reaction to justify a declaration whether or not heparin will be more satisfactory than other anticoagulants.

It is important, however, that the incidence of reactions from transfusion be minimized, because Sibley and Lundy have demonstrated that, on the tenth day after transfusion, the increase in total hemoglobin of a group of patients who had no reactions was 50 per cent more than the increase attained by patients who had reactions.

COMMENT

When small children are to be given transfusions of blood, the use of pentobarbital sodium[12, 14] (nembutal) in doses sufficient to keep the patient quiet will make the procedure easier. Apprehensive adults also

may be given pentobarbital sodium prior to transfusion with definite advantages. Further suggestions to facilitate venipuncture have been published elsewhere.[10]

The indications for blood transfusion are many, but some of the most common indications are the presence of one or more of the following: (1) shock owing to loss of circulating blood, (2) hemorrhagic diseases and blood dyscrasias, (3) debility with or without secondary anemia. Transfusions of from 1,000 c.c. to 5,000 c.c. of blood are not uncommon and are particularly indicated in shock owing to loss of blood and tissue fluid. When such large amounts of blood are administered, it is best to insert into the vein a needle which has a stylet. Then the slow drip of blood can be stopped for periods of one-half hour and the stylet can be inserted and left in place until it is desired to resume the transfusion. By this method the same needle and vein can be used for two or three days. Sometimes gold-plated needles are used for such prolonged transfusions because there is less tissue reaction at the site of venipuncture. Immunotransfusions are being used more extensively than before in cases of anterior poliomyelitis, measles, scarlet fever, and various forms of septicemia, but, as yet, there is no volume of evidence to make the procedure standardized. In certain cases, however, immunotransfusions have been remarkably helpful.

Another source of blood which I have not mentioned, but which ultimately may be used extensively, is the placenta. The method has been described by Goodall and others and by Griffith. Briefly the technique is as follows: After delivery of the child, the umbilical cord is sterilized with 5 per cent solution of iodine and cut. The end of the cord, which is still attached to the placenta, is placed in a sterile flask and the blood from the placenta is collected in this flask which contains a preservative solution. It is possible to collect between 100 c.c. and 150 c.c. of blood in the average case. Specimens of the placental blood are taken separately for blood grouping and serologic determination. When these data are available, the blood can be pooled in larger flasks for use when indicated. It has been shown that placental blood contains about 50 per cent more cellular elements and has 20 to 30 per cent greater coagulating power than has systemic blood of adult human beings. This method of transfusion is obviously important to certain obstetric and pediatric clinics and hospital services.

SUMMARY

Professional or nonprofessional donors can be used for blood transfusion. The blood may be given directly or indirectly and, if the latter method is employed, the blood may be stored and used as needed. The technique of indirect transfusion is not difficult if a simplified apparatus is used.

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Editorials

Pathologic Physiology and Diagnosis of Jaundice

IN THE study of jaundice there has often been confusion of two topics which are clearly distinct; i.e., (1) the site of formation of bilirubin and (2) the mode of development of jaundice.[1] In other words, simple overproduction of bilirubin, whether it occurs in the Kupffer cells of the liver or in other reticuloendothelial or mesenchymal cells, is not synonymous with jaundice.

Rich[2] classified the various forms of jaundice in two groups, depending upon which of two fundamental abnormalities was present. In "retention" jaundice bilirubin is supplied to the circulating blood more rapidly than the liver cells are able to remove it; other bile constituents, however, are not accumulated, and probably as a result (although exact information is still needed on this point) the van den Bergh reaction is delayed or indirect in type, and bilirubin is not found in the urine regardless of the degree of hyperbilirubinemia. In "regurgitation" jaundice it is probable that whole bile is returned to the blood stream; in consequence the van den Bergh reaction is direct in type, and bilirubinuria is observed at relatively low levels of hyperbilirubinemia (usually at concentrations above 2 mg. per cent).

The clinical applicability of Rich's classification is made somewhat difficult by the frequency with which "retention" and "regurgitation" factors are present in the same individual. Hepatocellular injury is obvious in cases of jaundice due to diffuse parenchymal liver damage, such as that occurring in acute or subacute atrophy, cirrhosis, chronic passive congestion, neoarsphenamine jaundice, and with various severe infections. It might be supposed that the jaundice in these cases was solely "retentive," and in the early stages or milder degrees it appears that this is often true. Thus in cardiac jaundice, likewise in that due to arsphenamine, the van den Bergh reaction is often for a time delayed or indirect, and bilirubin is not demonstrable in the urine, but as the severity of the condition increases the van den Bergh reaction becomes direct and bilirubinuria appears. The experimental studies of Hiyeda[3] and Itoh[4] have shown that the "regurgitation" of bile in instances of diffuse liver damage, such as that due to toluylendiamine poisoning in dogs, is due to injury of the terminal portions of the bile capillaries with resultant increase in permeability. The ensuing jaundice was shown to be due to "diapedesis" of bile into the surrounding lymphatic

spaces. This type was contrasted with the jaundice due to obstruction of the larger bile ducts, and again it was found that the leakage of bile occurred from the terminal or ampullary portion of the bile capillaries; in this type, however, regurgitation of bile was due to actual "rhexis" of the bile capillary wall as a result of the gradually increasing intrabiliary pressure. It is of significance that the end-result in both types is much the same; i.e., escape of bile into the lymphatic spaces, thence to the thoracic duct and finally the circulating blood. Thus, while there are probably two modes of regurgitation of bile, the end-result is the same so far as concerns the appearance of a direct van den Bergh reaction and of bilirubin in the urine. From the clinical viewpoint, especially the question of prognosis and the desirability of surgery, it is clearly not enough to determine simply that the patient has "regurgitation" jaundice. In other words, the qualitative van den Bergh reaction has not proved of value in the clinical distinction of jaundice due to diffuse parenchymal liver damage from that due to obstruction of the larger bile ducts. The qualitative van den Bergh test has proved of chief value in the distinction of "hemolytic" jaundice, which represents the most often encountered member of the "retentive" type. It should be emphasized that the indirect or purely delayed van den Bergh reaction, although a regular finding in hemolytic jaundice, is not to be regarded as pathognomonic of this disease. As noted above, this type of reaction may be observed in various forms of liver dysfunction of mild degree. One condition most readily confused with hemolytic jaundice, if the van den Bergh reaction alone is the diagnostic criterion, is the relatively rare "constitutional hepatic dysfunction,"[5] or simple cholemia of Gilbert. This affection simulates familial hemolytic jaundice in the mild degree and long duration of the icterus, in being "acholuric," and in exhibiting an indirect or purely delayed direct van den Bergh reaction. In other respects the conditions are quite dissimilar; splenomegaly, increased reticulocyte percentage, fragile microcytes, and increased excretion of urobilinogen in the feces are all lacking in constitutional hepatic dysfunction. It is well to point out that urobilinogen may or may not be present in excess in the urine in either condition. Increases are by no means always encountered in hemolytic jaundice, as has often been believed. Cases of hemolytic jaundice with little or no increase of urobilinogen in the urine also have less retention of bilirubin in the plasma. At times there may even be a total absence of jaundice in spite of a marked increase in the rate of hemoglobin wastage. In one case studied by the writer there had never been jaundice and the outstanding symptoms were splenomegaly and profound anemia. Examination of the feces revealed an increase in urobilinogen of about five times normal. The urine in this case did not contain any excess of urobilinogen. The blood exhibited fragile microcytes and an increased percentage of reticulocytes. Splenectomy was curative. It was clear

that liver function in this patient was entirely capable of keeping up with the increased pigment metabolism. The findings bear out the statement made at the beginning of this discussion; i.e., that increased bilirubin formation and the appearance of jaundice are not synonymous.

Of chief clinical importance is the differential diagnosis of the various forms of regurgitation jaundice, those which are characterized by bilirubinuria and direct van den Bergh reaction. From the standpoint of prognosis and management these may be classified in three groups: (1) calculous jaundice, (2) cancerous jaundice, and (3) parenchymal jaundice.

In the distinction of the three groups mentioned, the physical examination should be especially careful with relation to the gall bladder, which, if distended and palpable, points most strongly toward cancerous biliary obstruction; and to the spleen, which, if appreciably enlarged, is very suggestive of parenchymal jaundice. The size and character of the liver is important, large livers having little differential significance except if they contain distinct nodular metastases; small livers, however, being much more indicative of cirrhosis or atrophy, that is to say, of parenchymal jaundice. Ascites is often difficult of interpretation; if fluid obtained at the first paracentesis has the characteristics of an exudate, and particularly if it contains mucin by the acetic acid test,[6] the jaundice is much more likely to be cancerous. "Foetor hepaticus," the hepatic or amine odor,[6, 7] a peculiar somewhat musty and sweetish odor which may even pervade the entire room in which the patient lies, is almost pathognomonic of liver atrophy. In the writer's experience this odor has been encountered in but five of a series of about 200 cases of jaundice; acute or subacute atrophy of the liver was present in each of the five.

The depth of jaundice is by no means solely dependent upon the degree of biliary obstruction or the disturbance in bilirubin excretory function of the liver. In the presence of complete biliary obstruction the depth of jaundice is directly proportional to the rate of wastage of hemoglobin.[8] Associated blood loss results in compensatory slowing of this rate with consequent reduction of jaundice, since the supply of bilirubin is diminished and there is constant loss in the urine. A marked decrease in jaundice is often wrongly ascribed to a release of the complete biliary obstruction, when in reality it is due to decreased bilirubin formation as a sequel to gastrointestinal bleeding. If the urobilinogen excretion in the feces is determined in such instances, it will be found not to have increased.[10]

When the rate of blood destruction is actually increased and at the same time the bilirubin excretory function of the liver is seriously impaired, the accumulation of bilirubin in the plasma will obviously proceed more rapidly and the depth of jaundice will be correspondingly great. This is not infrequently the case in severe parenchymal jaundice,

such as acute or subacute atrophy, the more severe and prolonged cases of acute catarrhal jaundice, and in some instances of cirrhosis. When the depth of jaundice is marked because of an increased rate of blood destruction, the reticulocyte percentage will often be found elevated.

The degree of biliary obstruction or of diminution of bile flow is a matter of distinct importance in the differential diagnosis of the three groups of jaundice mentioned above. Information on this point can be obtained best by determination of the amounts of urobilinogen excreted in the feces.[9, 10] Because of the inconstancy of bowel movements and the varying dilution of the feces, it is necessary to determine the per diem excretion for a given period of time. In the writer's studies[9, 10] comparisons were based on the average output over a four-day period. It was found that cancerous jaundice was characterized by complete biliary obstruction, not more than 5 mg. of urobilinogen per day appearing in the feces and not more than traces in the twenty-four-hour urine. The term "cancerous jaundice" did not include cases of primary or metastatic carcinoma of the liver, in which jaundice of appreciable degree but rarely occurs. Up to the present time, 56 cases of cancerous jaundice have been investigated with respect to per diem urobilinogen excretion. In 53 of this group the per diem amount of urobilinogen was less than 5 mg., although in 1 of these cases the amount later increased to over 70 mg., an increase which was associated with local signs suggesting that an internal biliary fistula had become established. In 3 cases the amount was more than 5 mg. per day. One of these, with a carcinoma of the gall bladder involving the common duct, excreted 7.5 mg. and 7.2 mg. daily during the two four-day periods of study. In the other 2 cases there were polypoid tumors in the common duct which were responsible for incomplete obstruction; 1 of these followed over a considerable period of time, exhibited intermittent complete obstruction, and at operation it was clear that the polypoid tumor just above the ampulla of Vater had acted as a ball valve. In contrast with the cancerous group are the results in 49 cases of calculous jaundice and 62 cases of parenchymal jaundice, the latter series including acute catarrhal jaundice, acute atrophy, and cirrhosis. On the total of 111 cases of both groups, only 5 excreted less than 5 mg. of urobilinogen per day in the feces. In 3 of this number biliary obstruction was due to a common duct stone, while in 2 there was complete cessation of bile flow associated with liver disease. One of the latter had neoarsphenamine jaundice; the other had acute yellow atrophy of undetermined etiology. In this connection it may be noted that 5 cases of acute or subacute atrophy of the liver were included in the parenchymal group; in 4 of these the per diem urobilinogen was more than 5 mg. per day, in other words more than what may be spoken of as the "cancerous" range. Several cases of cirrhosis were encountered in which, despite an obvious

element of regurgitation jaundice, the amounts of urobilinogen in the feces were considerably greater than normal, a finding indicative of an increased rate of blood destruction.

The characteristic complete obstruction in "cancerous" jaundice is without doubt related to the infiltrating and stenosing effect of the neoplasm. On the other hand, it is believed that complete obstruction in calculous jaundice is but rarely encountered because of the constantly dilating effect of a common duct stone. Even when stones are impacted in the ampulla, the egress of small amounts of bile is usually permitted, and over a four-day period this is reflected in an increase of the per diem urobilinogen above the "cancerous" range.

Hemolytic jaundice is readily distinguished from each of the three above groups, by virtue of indirect or delayed van den Bergh reaction, absence of bilirubin in the urine, increased excretion of urobilinogen in the feces, and elevated reticulocyte count.

It is believed that an increase of urobilinogen in the urine, over the normal limit of 3.4 mg. in twenty-four hours, is evidence at least of temporary liver dysfunction. The increase is most marked and constant in instances of parenchymal jaundice, particularly those in which there is an associated elevation of the rate of blood destruction, such as in the cases of hepatic cirrhosis, or prolonged and severe catarrhal jaundice exhibiting macrocytic hemolytic anemia.[10] Considerable increases are not uncommonly observed in familial hemolytic jaundice; on the other hand, there is often no increase whatever even though a severe hemolytic process is present. There is much reason to believe that the increases which are seen in hemolytic jaundice are due to actual liver dysfunction, not to simple overflow because of the excessive formation of urobilinogen.[11] It should not be assumed that the urine urobilinogen is regularly increased in the parenchymal jaundice group. In occasional cases the reduction in bile flow may be so marked that the amount of urobilinogen returning to the liver becomes relatively insignificant. In these cases even ordinarily normal amounts must be evaluated; for instance, if the per diem output in the feces is but 10 mg., that in the urine 2 mg., the latter value would strongly suggest liver damage when the stool-urine ratio is taken into account. In some cases of parenchymal jaundice, particularly cirrhosis, the increase of urobilinogen in the urine is intermittent and may not be observed unless several twenty-four-hour determinations are made.

Prognosis and management of the jaundiced patient depends upon distinction of calculous, cancerous, parenchymal, and hemolytic forms. In calculous jaundice, operation should be deferred in the hope that the jaundice will recede, as it most often does. Operation can be carried out with considerably less risk after the jaundice has disappeared or greatly diminished. If the jaundice does not recede within two weeks (no increase in the feces urobilinogen and no decline of serum bilirubin),

then the common duct should be explored. Operation, if advisable at all, should be carried out earlier in cancerous jaundice since here the chances of release are almost nil. In parenchymal jaundice medical management alone is to be considered, with the exception of certain cases of hepatic cirrhosis associated with splenomegaly and macrocytic hemolytic anemia, in which splenectomy may be of value.[7] Splenectomy is usually desirable in cases of familial hemolytic jaundice.

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Regarding Practices Frequently Used for Prevention and Treatment of Postoperative Distention

COMMENT was made in a recent editorial upon the extent to which the profession retains empirical practices, frequently carrying useless or harmful therapeutic customs from decade to decade without proper scientific warrant. Particular reference was made to the unphysiologic practice of promiscuously giving postoperative enemas: "A patient who has abdominal distention and complains of gas pains is frequently treated by the administration of enemas and flushes. It is irrational to assume that the gastro-intestinal tract which is functionally inactive and already unable to empty itself of its contained fluid and gas will function normally after further overloading it by the administration of additional fluid in the form of an enema. Everything else

being equal, the degree, the extent, and the duration of post-operative distention and gas pains are directly proportionate to the number and the size of the enemas and the flushes administered, and yet how many physicians will allow a patient to go 1, 2, or even 4 days without having a bowel evacuation because the necessity of daily evacuation has been empirically used.''[3]

It is my belief that not only is the enema frequently used to the disadvantage of the patient postoperatively but that the following commonly practiced procedures also frequently predispose to postoperative distention and discomfort; and that these measures should not be used promiscuously and without indications of more clear-cut character than those commonly accepted: (1) the preoperative enema; (2) preoperative catharsis (in general abandoned at present but still practiced to some degree and extent); (3) preoperative and promiscuous postoperative administration of so-called specific ''gut toning'' drugs; (4) insertion of rectal tubes of undue dimension for relief of ''gas pains''; (5) unindicated bladder catheterization; and (6) routine proctoclysis.

The mechanisms which control gut tone, the exact distribution of sympathetic and parasympathetic endings in parts of the gut wall, the methods of reactions of the nerve endings and the musculature itself to different stimuli are in some respects controversial points with physiologists and neurologists. No definite evidence can be offered at present that predisposition to distention may *not* occur from improper use of the measures just cited; whereas, some logical experimental and clinical experiences indicate that these measures may predispose to distention.

What sound physiological evidence has ever been produced to show that harm will arise from a normal amount of fecal matter lying in its normal habitat during and for a short time after a routine operation? Is it not more physiologic to leave it so since it is normally present, since perhaps it might in itself by mechanical or chemical means assist in maintaining the normal gut tone? Hewlett, in speaking of lack of normal stimuli to the gut, says: ''The transportation of material through the intestinal canal is governed chiefly by the action of the intestinal contents upon the local nervous mechanism and muscle fibers of the surrounding intestinal wall. The stimuli which exert this action may be of a chemical or mechanical nature. Among the chemical substances that normally influence the movements are certain products such as carbon dioxide, marsh gas, skatol, etc.''[2] Attempts to remove fecal matter in the colon preoperatively may result not only in a mildly irritating solution but also in variable amounts of air being frequently introduced as well. And undoubtedly both parts of the enema solution and introduced air are frequently retained to be absorbed or possibly to produce reflex disturbances in the entire intestinal tract.

Alvarez showed that pieces of muscle excised from the gut wall of a purged rabbit were more irritable than normal in some instances and

that in others the segments would hardly respond to stimulation at all.[1] Though expulsion of the enema, we assume, is prompted chiefly by stretching the smooth muscle, it would seem that a soapsuds enema may be as irritating at times to the mucosa of the colon as a cathartic, thereby possibly setting up mucosal reflexes which, allied with muscle stretching, might create, after the enema, more initial spasticity followed by inactivity and diminished tone than is normally present.

For the past six years, we have avoided the promiscuous use of both the preoperative and postoperative enema, ordering it only in special instances where, for example, surgery upon the colon was anticipated or when especially indicated postoperatively. Since adopting this routine, it definitely seems that postoperative distention in our patients has been less frequent and severe.

Surely a consideration of the fundamental laws of intestinal physiology, some of which have been discussed above, together with an appreciation of the transient action of at least some stimulants commonly recommended for prophylaxis against postoperative distention, would indicate that the routine prophylactic administration of such drugs is fallacious therapy. It seems logical to assume that preoperative stimulation of a normally functioning gut in an attempt to increase its tone postoperatively is not only to stimulate needlessly the mechanisms concerned in maintaining the tone but thereby possibly to predispose to greater relaxation and later distention through this needless stimulation. Morphine as usually administered procures, if the work of Ochsner and the observations of certain physiologists are to be accepted, a mild and more desirable prophylactic segmental stimulation of the gut than is to be obtained with extracts of the pituitary gland.

Colon or rectal tubes of large size should not be inserted in the rectum to allow passage of gas through unduly spastic sphincters. An ordinary urinary catheter sufficiently new that the walls will not be collapsed by the sphincters certainly has a lumen amply large for passage of gas. Only those who have had large rectal tubes inserted and compared with ordinary No. 16 F catheters can appreciate the contrast in comfort between the two tubes. It often is not sufficiently appreciated how much tiresome discomfort a sensitive patient may obtain from the sphincters being dilated by a large colon or rectal tube which is allowed to remain within the rectum over varying periods of time. Unnecessary stretching of the sphincters through afferent impulses causes reflex stimulation of sympathetics, thereby possibly predisposing to inactivity and distention of the bowel.

Laws regarding the sensitivity of reflex action may be applied to unnecessary catheterization. Hewlett says, in speaking of intestinal tone: "Nervous reflexes may diminish the intestinal movements as has been shown by experiments on animals (compression of the testicles). . . ."[2]

Urinary catheterization is a painful procedure to some people. It is not proper here to enter in a discussion regarding when catheterization should and should not be done. An unduly distended bladder in postoperative patients is undesirable, but we do believe that catheterization is ordered routinely more frequently than it should be.

Regarding routine proctoclysis only a few words will be said. A proctoclysis is, in a sense, a retained enema. Through mechanisms discussed above, may it not frequently be a source of postoperative discomfort not only directly but secondarily by reflex disturbance of the intestinal tract, predisposing to stimulation of sympathetics and distention? It should not be used routinely; only on special indication.

All in all it would surely seem, on consideration of sound physiologic principles, that it is a common tendency to tamper entirely too much with the intestinal tract pre- and postoperatively; and our clinical experience would indicate that the incidence of postoperative distention may be decreased by a more general consideration of relevant physiologic laws.

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Recent Advances in Surgery

CONDUCTED BY ALFRED BLALOCK, M.D.

OBSTRUCTIVE JAUNDICE

A CRITICAL REVIEW

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ALTHOUGH jaundice is merely the clinical evidence of an underlying pathologic process and is not a pathologic entity in itself, its appearance is so significant and may be so serious that it has come to be regarded and discussed almost as if it were a disease. In spite of the very valuable contributions to the subject which each year brings forth, jaundice still presents many serious and unsolved problems. Within the last few years a few almost revolutionary facts have been revealed concerning so-called obstructive jaundice, the type with which this review is concerned, and there is a continued emphasis upon other and older facts and theories.

CLASSIFICATION

It is now generally accepted that jaundice will occur whenever there is a disturbance of balance between the amount of bilirubin delivered to the liver for excretion and the capacity of the liver to excrete the pigment delivered to it. At that point, however, agreement ends, and this is particularly true of the classification of jaundice, a consideration which is of basic importance in any discussion of the condition.

The classification proposed by McNee[1] in 1923 was a distinct advance over anything which had preceded it, and it is still in very general use because it is simple and convenient. It includes obstructive hepatic jaundice, which implies mechanical interference with the flow of bile through the extrahepatic channels; toxic or infectious jaundice, which implies a block in the hepatic filter, the result of toxic or infectious injury of the hepatic cells, and hemolytic jaundice, which implies an increased rate of destruction of the red blood cells and a resulting production of bilirubin beyond the excretory capacity of the liver to care for.

In 1933 McNee,[2] defending this classification before the Royal Society of Medicine, pointed out that it was almost universally misquoted be-

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cause the word hepatic was almost universally dropped from the first two groupings, an omission, he went on to say, which carried with it a complete loss of emphasis upon the role of the liver in the production of jaundice. He declared further that his classification had helped to change Minkowski and Naunyn's original statement, "Ohne Leber, kein Ikterus" (without the liver, no jaundice), into Aschoff's later statement, "Ohne mangelhafte Ausscheidung des Gallenfarsstoffs durch die Leber, kein Ikterus" (without defective excretion of biliary pigment via the liver, no jaundice).

McNee's apologia is true as far as it goes, but it does not go far enough. It does not overcome the fundamental misconception of the classification, natural enough when it was advanced, which is the postulate that the bilirubin in the blood differs in different varieties of jaundice. That idea was completely overthrown a few years later by the work of Harrop and Barron,[3] which is excellently summarized by Rich,[4] about as follows:

Pure bilirubin at the pH of the blood gives the direct or immediate van den Bergh reaction, but in normal plasma it gives the indirect reaction, because of the absorption of the bilirubin by the plasma proteins; as a result, the pigment is prevented from reacting promptly with the reagent. When, however, substances such as bile salts and cholesterin, which are present in small quantities in whole bile and in increased quantities in the plasma in the various forms of jaundice, are added to the plasma before bilirubin is introduced, or are added at the same time as the bilirubin, the van den Bergh reaction remains direct, apparently because substances which lower surface tension seem to be adsorbed by the proteins more readily than bilirubin is adsorbed. When the protein molecules are covered with such substances, the pigment remains free.

In short, experimental and clinical studies have made it clear that, when the plasma bilirubin of a jaundiced subject is tested by the van den Bergh method, the direct reaction may be taken to indicate that whole bile, which contains bile acids and cholesterin as well as bilirubin, has been regurgitated into the blood stream. It may further be assumed that the underlying pathologic process is either an obstruction of the bile ducts or necrosis of the liver cells, for these are the two conditions which permit bile to escape from the canaliculi into the blood stream. On the other hand, the indirect van den Bergh reaction may be taken to indicate that the bilirubin in the plasma has not been regurgitated into the blood from the canaliculi but represents pigment which the liver has not been able to remove from the blood stream. Obviously, then, the underlying pathologic process is an overproduction of bilirubin or a depressed excretory function of the liver, or a combination of these two states. The biphasic van den Bergh reaction can occur as the result of a high degree of pigment retention, without regurgitation, or under certain

circumstances of biliary regurgitation caused by obstruction or by necrosis of the liver cells.

On the basis of this work, Rich proposed a new classification of jaundice into the retention and the regurgitant varieties. In the retention type the bilirubin circulating in the blood stream is only partially excreted by the liver. The remainder accumulates in the blood, where it is bound, probably to the plasma proteins, and therefore does not pass through the kidneys into the urine. This type of jaundice is associated with mild forms of liver injury, chiefly cloudy swelling and cell atrophy, and is a secondary complication of such diseases as hemolytic anemia, congestive heart failure, pneumonia, and other diseases which are characterized by anoxemia and fever. In such diseases the jaundice is due partly to an increased formation of bilirubin and partly to a depressed excretory function of the liver. The van den Bergh reaction is indirect.

In the regurgitant type of jaundice the bilirubin circulating in the blood stream is excreted by the liver cells and escapes into the tissue spaces and blood sinusoids of the liver because of the rupture of the bile canaliculi. The larger bile ducts are obstructed, or the liver cells are necrotic, or both conditions occur in combination, and, as a result, both bilirubin and whole bile escape into the blood stream. The bile pigment is not bound and therefore can be excreted by the kidneys into the urine. This type of jaundice is associated with the major hepatic disorders and with mechanical obstruction of the extrahepatic bile ducts. The van den Bergh reaction is direct.

Although the classification advanced by Rich undoubtedly rests upon a sounder basis than the earlier classification of McNee, it is rather generally ignored. Because it requires for its comprehension and employment a more exact knowledge of the fundamental pathology of jaundice, many writers state frankly that the average practitioner is better equipped to use the classification of McNee. Convenience and simplicity, however, do not compensate for unsound premises. Rich's classification has no therapeutic intent and should not be condemned on that basis, as it very often is. Furthermore, although obstructive jaundice, strictly speaking, includes both the intrahepatic and the extrahepatic varieties of obstruction, common usage has so long attached to the term the limited implication of mechanical extrahepatic occlusion that it will probably continue to be used in that sense without serious confusion. It is so used throughout this paper.

Elton[5] advances a "working hypothesis" for the mechanism of jaundice, based on the premise that the forms of bilirubin differ qualitatively. By his theory, bilirubin, as it passes through the liver, is changed to a water-soluble, crystalloid bilirubinate, which gives a direct van den Bergh reaction, while the unaltered form gives an indirect reaction. It seems far more reasonable to accept the work of

Harrop and Barron and to explain the direct and indirect van den Bergh reaction by the presence or absence of bile salts in the blood. Elton's division of the liver into major functional zones, the acceptance zone and the excretion zone, and an intermediate conversion zone, seems unsupported by the evidence now available.

EXPERIMENTAL AND GENERAL

The comprehensive review of the experimental aspects of jaundice which was published by Mann and Bollman[6] in 1935 still remains the most valuable single contribution to this phase of the subject. Regardless of how obstructive jaundice is produced, they point out, its results are due to three factors; namely, the effect on the body cells of the retention of bile in the blood and in the tissue fluids, the effect of the absence of bile or the alteration of bile in the gastrointestinal tract, and the effect of the hepatic injury which accompanies jaundice. Complete loss of bile is compatible with life for an indefinite period of time if adequate care and a suitable diet are maintained. Nutritional disturbances are due to loss of normal bile from the gastrointestinal tract, plus the effect of retention of bile. The anemia and the diminution of plasma proteins are due partly to dietary and absorptive factors and partly to associated hepatic injury. An important clinical consideration is pointed out in this connection: These findings suggest the addition of large amounts of meat to the diet, but actually the jaundiced animal does not tolerate such a regimen over any period of time. Life can be prolonged indefinitely, however, on a diet which includes milk and syrup. Ascites may occur under conditions in which it does not usually occur in the nonjaundiced animal. Finally, bile salts continue to be formed, though in somewhat reduced amounts, until increasing hepatic damage entirely prevents their formation.

Walters and his associates[7, 8] have contributed valuable discussions on the pathologic physiology of stone in the common duct, and Snell[9] has studied the same condition in relation to the structure and functions of the liver. After classifying into four groups the sixty-three cases which form the basis of his study, he concludes that in two-thirds of such patients the hepatic lesions are transient and relatively minor, and offer no great obstacle to recovery. The remaining third includes the cases which are more serious and which exhibit various degrees of degenerative or destructive lesions of the parenchyma, proliferation of connective tissue and cirrhosis, and cholangiectasis and cholangeitis, all associated with disturbances of hepatic function.

Rozendaal, Comfort, and Snell,[10] in a study of 138 individuals without hemolytic disease, found increased concentrations of bilirubin which they took to indicate hepatic dysfunction, either constitutional or secondary to hepatic injury. Such patients are prone to "bilious attacks" and exhibit intermittently or even constantly a mild degree of

jaundice which may later terminate in definite cholecystic disease, as happened in 29 per cent of the cases in this series. The authors note pessimistically that many of the other patients in the group are young and may later fall victims to such disease. They recommend a wider use of the bilirubin test both to demonstrate individuals likely to develop cholecystic disease and to reveal unsuspected cases of mild functional and organic disturbances of the liver.

This conception is of special interest to those who believe, as we do,[11, 12] in Henschen's[13, 14] theory of "liver weaklings." Our own conception of the so-called liver death is that certain individuals whose hepatic function is adequate for the stress and strain of ordinary life cannot withstand the added strain of surgery and all that it implies, and lose their lives because no provision is made for their "weak spot." We[15, 16] have been using the Quick[17,18] hippuric acid liver function test in the preoperative study of many types of surgical subjects, and have found a rather surprising number of individuals who reveal an entirely unsuspected hepatic dysfunction but who have responded well to preoperative preparation. Such preparation is omitted in the usual case because the necessity for it is not evident, and that omission, we believe, is responsible for the occasional tragedy that follows simple elective surgery in presumably safe surgical risks. This conception is in line with Graham's[19] repeated warning that individuals with damaged livers are questionable subjects for any sort of surgery.

It should also be remembered that, as Rozendaal's study shows, the existence of jaundice is apparently not incompatible with reasonably good health or even with perfect health.

ETIOLOGIC FACTORS

There is little new in the literature dealing with the etiologic factors of jaundice, although many reports testify to the unwisdom of fixed theories concerning their frequency. Thus, Heyd[20] lists in the order of frequency gallstones and infection of the extrahepatic bile ducts, catarrhal jaundice, hepatic degeneration, cancer of the liver, cirrhosis of the liver, cancer of the bile ducts or gall bladder, cancer of the pancreas, and metastatic carcinoma. Eusterman,[21] on the other hand, lists gallstones, cancer of the pancreas, stricture of the extrahepatic bile ducts, congenital and acquired hemolytic jaundice, intrahepatic jaundice, primary neoplasms of the extrahepatic bile ducts, cancer of the gall bladder, acute and chronic pancreatitis, noncalculous cholecystitis, and a miscellaneous group, including general carcinomatosis, metastatic carcinoma, cholangeitis, and primary carcinoma of the liver and duodenum. The general experience is that gallstones are the most frequent cause of obstructive jaundice, as in both these studies, but that carcinoma of the pancreas is a far more frequent cause than carcinoma of the liver, the bile ducts, or the gall bladder. Walton[22] notes that his

personal incidence of carcinoma of the gall bladder (31 cases), as opposed to his personal incidence of carcinoma of the bile ducts (33 cases), is contrary to the usual figures, which show the latter condition to be twice as infrequent as the former.

Elton[23] studied the frequency of jaundice in a representative hospital population by means of a two-month survey of all the patients admitted to St. Mary's Hospital in Detroit. The incidence was 1 in every 8 patients, but in one-third of the cases the icterus was simply associated with interstitial blood extravasations following accidental or surgical trauma. A rather surprising range of conditions was revealed in the remainder of the cases. The study involved the use in all cases of the icteric index, the van den Bergh reaction, and the quantitative serum bilirubin test, and the author points out that failure to use the three tests in combination often will give rise to confusing and misleading conclusions, as will failure to use the proper standard (0.25 mg. per 100 c.c. of blood) for the serum bilirubin test.

There is an increasing emphasis upon noncalculous lesions of the ducts as a cause of obstructive jaundice. Ellsworth Eliot's[24] study of benign cicatricial strictures is perhaps the most complete available, but excellent reviews have also been contributed by Walters[25] and by Lahey.[26] Ladd[27] collected, from the records of the Boston Children's Hospital, 45 cases of congenital obstruction of the bile ducts, which he classified into seven distinct groups, and Donovan[28] collected 18 cases from the Babies' Hospital and St. Luke's Hospital in New York. Ladd considers that 37 per cent of his cases presented a possibility of cure, and his ultimate results suggest that choledochoduodenostomy is preferable to cholecystduodenostomy. Donovan's outlook is more pessimistic, though, like Ladd, he emphasizes the importance of exploration in even the most hopeless-seeming cases. He points out also that the findings at operation may be incorrect, for two of his patients, in whom obliteration of the ducts was thought to be complete, were alive and well two and one-half and three years respectively after operation.

Diffuse contracture of the extrahepatic ducts, according to Ransom and Malcolm,[29] is hopeless if all the ducts are involved, though hepaticoduodenostomy may be useful if the hepatic duct is not occluded, and cholecystenterostomy is justified if the gall bladder contains bile. Patterson[30] describes a case of jaundice due to obstruction of the common duct by an aberrant vessel, and another case, which Graham[31] is not inclined to accept, due to adhesions. Nicholson[32] reports a case of jaundice associated with diverticulum of the duodenum and analyzes the 14 cases reported in the literature. Cystic dilatation of the common duct as a possible cause of jaundice is mentioned by Walton, who has personally seen 2 cases, and by Taylor,[33] who collected 4 cases, 2 of which were observed personally, over a ten-year period at the University Hospital in London. Pavel[34] describes functional obstructive jaundice due to

reflex spasm at the common duct sphincter. Medical treatment by the Einhorn tube is sometimes successful, but more often surgery must be resorted to, and the deviation of the bile must be carried out over a long period of time. His statement that surgery is without danger (from hepatitis) in long-standing jaundice indicates a casual point of view not likely to find general acceptance.

Potter[35] collected from the literature and from his personal records 432 cases of biliary tract disease in young subjects (under 15 years of age), in 209 of which jaundice was also present. Since youth is generally regarded as a point against the diagnosis of obstructive jaundice, these figures should be borne in mind. Penberthy and Benson[36] have called attention to the same possibility.

DIAGNOSIS

Eusterman's study of 533 cases of jaundice verified by operation or autopsy remains the best single study of the diagnostic errors which are possible in this condition. There will be general agreement with Ottenberg and Colp's[37] conclusion, based on a careful analysis of 84 cases, that no single sign, symptom, or test can be relied upon for diagnosis, just as all experience bears out Lord Moynihan's statement, now many years old, that no man living is infallible in the diagnosis of obstructive jaundice.

Eusterman lists as possible sources of error: the absence of any trustworthy specific test to differentiate the varieties of jaundice, especially when obstructive jaundice is painless or incomplete; the simultaneous occurrence of several lesions in the biliary tract of the same subject; the existence of pathologic conditions with which it is unusual to associate jaundice; and the existence of conditions difficult to recognize clinically which may produce mechanical jaundice. Ottenberg and Colp list as points of diagnostic value: pain, chills, a history of previous similar attacks, a history of the ingestion of toxic substances, the presence of a palpable spleen or of local rigidity, the occurrence of fever at the onset, and a group of laboratory tests upon which we shall comment later.

Ottenberg and Colp also warn against reliance on the presence or absence of pain to differentiate obstructive from nonobstructive jaundice, or to differentiate benign from malignant conditions. Rives, Romano, and Sandifer[38] make the same point in their analysis of 96 cases of carcinoma of the pancreas, in 48 of which pain was associated with the jaundice. Rhodes and Greenblatt[39] have called attention to this source of error in their study of 24 cases of carcinoma of the gallbladder, and we have noted it in our own study[40] of 25 cases of the same condition and in another study[41] of 26 cases of carcinoma of the liver, in a small group of which pain and jaundice were associated. It must not be forgotten, too, as many writers point out, that gallstones may be present

along with a malignant process and may confuse the picture by the interjection of a type of colicky pain not supposedly associated with malignancy.

LABORATORY STUDIES

Martin's[42] point is well taken that the biochemical study of jaundice has become almost too unwieldy to be useful and that abstinence from unnecessary tests is a desideratum in patients who are frequently so ill that they greatly appreciate being left undisturbed. One cannot, however, practice that precept too literally, for laboratory studies, and especially serial laboratory studies, often furnish information which is essential and which cannot be dispensed with.

Laboratory tests in jaundice fall into two groups, those which are employed to demonstrate the jaundice or to differentiate the varieties, and those which are designed to study the associated liver damage. In the former group the value of the icteric index, the serum bilirubin test, and the van den Bergh test is so generally accepted that no comment upon them is necessary. Certain of the newer and less generally employed tests should be mentioned briefly.

Opinion differs as to the value of the galactose tolerance test. Snell and Magath,[43] in an excellent review of such tests, state that it furnishes helpful but not altogether reliable information as to the possible hepatogenous or obstructive character of the jaundice, and Ottenberg's[44] opinion is much the same. White[45] considers that a positive test (showing 6 to 7 gm.) is practically conclusive evidence of liver degeneration, which is not, however, excluded by a negative result. Schiff and Senior[46] believe it to be very valuable in differential diagnosis. In a study of 100 cases of jaundice they found the test negative in all 20 cases of obstructive jaundice, but positive in 49 of 50 cases of catarrhal jaundice and in 14 of 15 cases of toxic hepatitis.

Ottenberg advocates the serial use of the test for cholesterol and cholesterol esters. The work of Epstein and Greenspan[47] has shown that in biliary obstruction there is associated with the increase in the total blood cholesterol a parallel though somewhat less marked increase in the percentage of cholesterol esters. Ottenberg, who advocates the serial use of the test, points out that the tendency in all forms of hepatic degeneration is for the proportion of esters to fall, sometimes to within the lowest range at which accurate determination is possible by present methods; whereas, in obstructive jaundice the fall does not occur until the terminal stages, when extensive hepatic degeneration has occurred. In the early stages of obstruction an increase in the values is not unlikely.

The reliability of the serum phosphate test seems debatable. Cantarow and Nelson[48] found such a wide overlapping of values in a study of 31 patients with obstructive jaundice and 22 patients with nonobstructive jaundice that they consider the test worthless for differential diagnosis.

In the same issue of the same journal Flood, Gutman, and Gutman[49] report that the test is useful in ruling out obstructive jaundice, an opinion which is shared by Rothman and his associates.[50]

Ottenberg advocates the use of the Lichtman[51] test for tyrosine in the urine. Tyrosinase is a vegetable enzyme, derived most conveniently from potatoes, and capable of specifically oxidizing tyrosine to produce a dark brown pigment, melanin, which lends itself readily to colorimetric estimation. A positive test is of diagnostic value; a negative test means little. Small amounts of tyrosine in the urine of jaundiced patients suggest subacute degeneration of the liver, or malignancy, while large amounts point to acute liver autolysis. Mild degrees of tyrosinuria may occur in catarrhal jaundice. The value of the test from the standpoint of differential diagnosis is not yet established.

Jacobi[52, 53] has suggested that the glucose tolerance test be used to differentiate obstructive from nonobstructive jaundice and benign from malignant disease. In the obstructive variety of jaundice, he points out, the curve is still elevated at the end of a two-hour period; whereas, in the nonobstructive variety it falls promptly after the initial rise. When the test is repeated after the patient has been treated for two or three weeks with liver extract, a high carbohydrate diet, and large quantities of glucose, the curve is still elevated in malignant disease, but in benign states it assumes the character of the nonobstructive curve and falls sharply. The test is not yet in general use.

Foote and Carr[54] advocate the differential diagnosis of jaundice by fractional cholecystography. The dye is given intravenously, in divided doses, in combination with glucose, and radiography is repeated at intervals. Their latest report[55] covers 5 experimental and 15 clinical cases. By this method they believe they can say with assurance that, when the normal gall-bladder shadow is present at twenty hours but absent at forty-eight hours, intrahepatic disease is the cause of the jaundice; when the gall bladder or common duct is visualized at twenty hours and has not emptied at forty-eight hours, there is obstruction at or near the ampulla of Vater. They are unwilling to speak positively of the cases which show no shadow at twenty or forty-eight hours. Hunt, discussing the report, made the point that the method seems more exact in the experimental animal than as a clinical test, and questioned the wisdom of determining a therapeutic course on such tenuous evidence. It should be noted also that many experienced surgeons believe that the intravenous use of a dye, even when bolstered with glucose, is an unwise procedure in jaundice.

Sjostrom[56] has devised a test for the citric acid content of the blood serum, which, as Graham comments, eliminates the difficulty just mentioned. It is based on the premise that the liver cells contain an enzyme especially directed toward the citric acid metabolism, which

is damaged in hepatitis and also, though to a less marked degree, in jaundice due to stasis. In a series of 162 cases of diseases of the liver and bile ducts there was a prompt increase in the citric acid content of the blood serum in hepatitis, catarrhal jaundice, and cirrhosis, although the values remained normal until late in the disease in cholecystitis, cholelithiasis, and common duct obstruction. High values in these latter conditions indicated severe liver damage and presaged a high mortality. Graham's opinion is that the test seems of value in the differential diagnosis of jaundice and is worthy of an extended trial.

Von Purjesz[57] has described a simple method of demonstrating bilirubin in the urine, based on the oxidation of bilirubin to biliverdin by the use of sulfosalicylic acid and hydrogen peroxide. If bilirubin is present, a green coloration appears within fifteen minutes, ranging from olive green for small amounts to grass green for large amounts. The author claims that the test is both sensitive and reliable, and its simplicity should commend it to practitioners who lack elaborate laboratory facilities.

Malamud[58] studied the elimination of water in jaundiced patients by Volhard's test for induced diuresis. When normal patients are given a fixed amount of tepid, slightly sweetened, lemon-flavored tea, the urine voided in four hours is equal to or slightly in excess of the quantity of fluid ingested. In 18 of 20 jaundiced patients whose renal function was normal by standard tests, the quantity of urine voided over the four-hour period was markedly or slightly less than the fluid ingested, the degree of retardation depending upon the intensity of the jaundice. Other than demonstrating the effect of jaundice on the renal function, the test seems to have no clinical or prognostic value.

Brodribb and Cullinam[59] have devised a histamine test to demonstrate latent jaundice which cannot be demonstrated by the usual clinical or laboratory methods. When a minute quantity of histamine is injected intradermally, a deeply yellow wheal is raised if jaundice is present. Doubtful tests are regarded as negative. The histamine test was positive in 44 of 48 cases of obstructive and hepatogenous jaundice and negative in 22 normal controls; but, in spite of its apparent consistency, its limitations are obvious. The authors attribute the phenomenon to an abnormal permeability of the capillaries for bile pigment which is produced by the histamine and which allows the pigment to escape and concentrate in the wheal.

Mora and Jirka[60] used the Aldrich intracutaneous salt solution test in patients with obstructive jaundice and noted a marked diminution in the normal absorption time, with a prompt return to normal in 14 of 15 cases as the jaundice decreased. The explanation of the test is not clear.

We[15] have personally used the Quick[17, 18] hippuric acid test of liver function in a large number of jaundiced patients, and have found that it confirms the general idea that liver damage may or may not be asso-

ciated with the obstructive variety. It is interesting and very significant to trace the improvement in liver function after proper preparation for operation, as well as to note the fall in function after anesthesia and operation and the improvement as convalescence proceeds. Quick considers the test helpful in differentiating between obstructive jaundice, in which the values are likely to be normal or high, and catarrhal jaundice, in which they are likely to be markedly reduced. We should be unwilling to determine either diagnosis or therapy on this basis because of the many other factors which may confuse the values. Snell and Plunkett[61] share our views on this point, as well as our favorable opinion of the value of this test. They found a general agreement between the Quick test and the more expensive and elaborate serum bilirubin test. Kohlstaedt and Helmer[62] consider the extraction method described by Quick more accurate than the precipitation method in jaundiced patients, but we have found the simpler precipitation method entirely satisfactory.

DeCourcy,[63, 64] who studied the blood iodine in various types of biliary tract disease, found in 5 cases of common duct stone an increase from a normal of 3 to 6 μg to an average of 2,090 μg. Experimental studies substantiated his clinical findings. He considers operation reasonably safe when the blood iodine has fallen to 100 μg or less, and points out that this test is superior to the dye test of liver function because it does not require the introduction into the blood stream of a potentially harmful agent.

THE HEMORRHAGIC DIATHESIS

Walters'[65] experience is the experience of many surgeons, that 50 per cent of all deaths following surgery on jaundiced patients are due to hemorrhage. For some reason this is not the experience in the Charity Hospital of Louisiana at New Orleans. In a study of 100 deaths following biliary tract surgery we[66] personally found among 35 jaundiced patients only 4 deaths which could possibly be attributed to hemorrhage, and Rives, Romano, and Sandifer found only 4 such deaths in 72 cases of jaundice due to carcinoma of the pancreas. It is a curious and inexplicable coincidence that in two series of cases involving jaundice of totally different origins there should prevail a situation which is entirely contrary to general experience.

The defective clot theory to explain the hemorrhagic diathesis in jaundice is discussed by Ivy and his associates,[67] by Carr and Foote,[68] and by ourselves.[69] There no longer seems any doubt that the clotting mechanism in certain jaundiced patients differs from the normal mechanism, as can easily be demonstrated by simple observation. As we have pointed out, in patients with a hemorrhagic tendency the clot is large and bulky; it does not retract normally; it wets a large surface area of

filter paper when it is transferred to it; and it promptly falls to pieces when it is handled. The clot in a normal patient, or in a jaundiced patient without a bleeding tendency, is smaller, firmer, and definitely retractile, and it shows no tendency toward disintegration when it is handled. Observation of the clot is therefore a simple precaution which can be practiced by any surgeon in any case.

It is easy to explain the defective clot on the basis of Quick's[70, 71] studies. Both clinical and experimental evidence supports his theory of prothrombin deficiency as the most important, if not the only, cause of defective coagulation of the blood. Since the conversion of prothrombin to thrombin is a mass reaction, then a diminution in prothrombin necessarily is followed by a diminution in the production of thrombin, which in turn is followed by a diminution in the conversion of fibrinogen to fibrin, the essential element in a firm clot. A prothrombin deficiency is the fundamental cause, but a deficiency in the conversion of fibrinogen to fibrin is the immediate explanation of the character of the clot in patients with a bleeding tendency. Neither the bleeding time nor the clotting time is necessarily altered for a considerable period of time, regardless of the inadequacy of the mass reaction, for the conversion of fibrinogen to fibrin still occurs; the process is merely decreased; it is not eliminated. Indeed, Quick has shown that a critical level is not reached until the prothrombin content of the blood has fallen to 15 or 20 per cent. Further support of the theory of prothrombin deficiency is furnished by the experimental studies of Hawkins and Brinkhous[72] on bile fistulas.

Another practical consideration is the observation of Ivy and his associates that the tendency to hemorrhage is more likely to be evident twenty-four to forty-eight hours after operation than at operation, because during this period abnormally fragile clots are likely to be dislodged by postoperative vomiting, restlessness, and similar factors. We ourselves have pointed out that free oozing is more likely to occur at operation than hemorrhage, because the clot plugs the severed vessels, even though it does not plug them adequately. We also have emphasized a consideration which seems to be generally overlooked, the serious and treacherous character of capillary bleeding.

Snell[73] and Quick[71] recently have published very valuable summaries of the present knowledge of the hemorrhagic diathesis in jaundice. Three factors are associated with it. The first is the prothrombin deficiency demonstrated by Quick and his associates. The second is the vitamin K (koagulation) deficiency demonstrated by Dam[74] and by Almquist and Stokstad[75] and later confirmed by Quick,[76] who showed that, by the use of Almquist's diet, which is complete except for an adequate amount of vitamin K, a prothrombin deficiency was observed in chicks as early as the fourth day. When the prothrombin level

dropped below 20 per cent, a definite hemorrhagic tendency was invariably observed, though it could be promptly overcome by the use of small amounts of alfalfa, which is particularly rich in vitamin K. The work of Hawkins and Brinkhous and of Heyman[77] has shown, in addition, that in the absence of bile salts vitamin K is ineffective; their presence is clearly required for the normal absorption of fats and sterols from the intestine. Quick has also called attention to this point.

Many observers, Quick and his associates among them, are inclined to postulate that the prothrombin deficiency in the blood of certain jaundiced patients is the result of liver damage, and it must be granted that a large body of evidence, particularly the experimental and clinical evidence adduced by Quick in his most recent publication, goes to support that theory. The experimental studies of Roderick[78] and of Smith, Warner, and Brinkhous[79] also are in line with this conception. Logical as the reasoning is, however, we are personally still inclined to question an absolute cause and effect relationship between liver damage and the hemorrhagic diathesis; our personal studies[80] with Quick's own test of liver function have revealed a fair number of cases in which the hemorrhagic diathesis was evident and the liver function normal or approximately normal, though we must grant that we have also seen a few cases in which the bleeding tendency and a markedly deficient liver function were associated.

The gravity of the problem of the bleeding tendency in jaundiced patients, Quick points out, lies in the fact that its occurrence cannot be predicted with any degree of certainty by any method at all. As Ivy and his co-workers have remarked, the only way to determine whether a patient has a bleeding tendency seems to be to wait and see whether he is going to bleed. Various tests to determine this tendency have been devised. Carr, Foote, and Naffziger[68, 81] suggest the use of the Grote[82] test for sulfur compounds, on the theory that the clot in subjects with a hemorrhagic tendency is influenced by the collection of intermediate products of protein metabolism, such as cysteine and related forms of mercaptan. They profess satisfaction with the method in an extensive experimental study and a smaller series of clinical tests. We have not found this test reliable, and Ravdin and Riegel[83] question the role of sulfur compounds in the hemorrhagic diathesis.

In a small series of cases (7) Lewisohn[84] used an index arrived at by dividing by antithrombin the product of prothrombin multiplied by fibrinogen,[85*] any value below 0.7 indicating a tendency toward hemorrhage. In this series the test seemed accurate, but its requirements put it beyond the reach of the average practitioner.

Nygaard,[86] of the Mayo Clinic, studied the coagulation time of the plasma. In 238 normal subjects the mean was 190 seconds, against a

*Moss' study of fibrinogen is very valuable.

mean of 250 seconds in 90 jaundiced subjects. The plasma reading itself may be used, or an index arrived at by dividing the normal plasma coagulating time by the plasma coagulation time of the case in question; the normal index is 1. In the group Nygaard studied, the 6 patients who died of hemorrhage all had indices under 1.1. As we have previously commented, there seems no clear reason why an individual with an index actually higher than normal should exhibit a bleeding tendency, and the question may be properly raised as to whether the plasma clotting time parallels the coagulation time of the blood.

Ivy and his co-workers[67] have described a test for the hemorrhagic diathesis in which the factor of capillary retraction is eliminated and the burden of controlling hemorrhage is put squarely upon the factor of coagulation. The venous return in the upper extremity is cut off by the application of the cuff of a sphygmomanometer, with a pressure of 40 mm. of mercury, and a state is thus produced comparable to the clinical state produced by anesthesia, operative trauma, and shock, which in combination may result in capillary paresis and hemorrhage. In an extensive series of cases it was found that most jaundiced patients with a bleeding tendency showed a definitely prolonged venous pressure bleeding time, while most of those without such a tendency showed a venous pressure bleeding time within normal limits (four minutes).

This test has the virtue of simplicity and certainly should be used in all jaundiced patients in combination with other tests. It has, however, certain obvious defects. For one thing, the puncture is so minute that it is a question whether the equally minute clot is likely to be seriously affected by a limitation of the venous return. For another, the arbitrary upper limit (four minutes) for normal bleeding time permits the inclusion within it of patients with a bleeding tendency. We have also noted that the venous pressure bleeding time tends to remain low in younger persons and to become elevated with age. Finally, we[80] have sometimes found the Ivy test normal when our own serum volume test, to be described later, indicated a definite hemorrhagic tendency, which in one case, at least, was proved by actual bleeding. A recent report by Boys[87] corroborates our opinion of this test. In a study of 35 cases the 9 patients who bled showed an elevated Ivy bleeding time, but so also did 15 of the 26 patients who did not bleed. This seems a very high percentage of what might be called negative error.

The serum volume test for the hemorrhagic diathesis in jaundice which we[69] have described requires only a syringe and a graduated tube. An arbitrary amount of blood, preferably 3 c.c., is collected and allowed to stand at room temperature for four hours. The clot is then removed and studied and the serum volume read. The index is determined by dividing the serum volume by one-half the volume of blood withdrawn. The standard of normal is 1, and indices below this level are progressively

indicative of hemorrhage. The test is repeated as is necessary to determine the safe time for operation.*

A routine blood count is necessary to demonstrate anemia, for which the proper correction must be made; when it is present, the serum volume will naturally be greater than one-half the blood volume and the results of the test will be misleading. Also, the blood must be transferred quickly from the syringe to the tube, for the clot is defective if there is undue delay in this maneuver, and an unnaturally small amount of serum is expressed.

PREOPERATIVE THERAPY

Glucose still remains the sheet anchor in the preparation of jaundiced patients for operation, and the work of Althausen[88] on the value of its oral administration is worth careful consideration. Calcium also should be used, as suggested experimentally by Wright[89] and by Lee and Vincent,[90] and as applied clinically by Walters.[91] It has been clearly proved that no calcium deficiency exists in such cases, either actual or functional, but such therapy has undeniably good results, perhaps, as Ravdin's[83] studies seem to indicate, because of its effect on the liver.

The beneficial effects of blood transfusion are also clearly proved. Quick[71] has shown that the value of this therapy is due to a replacement of prothrombin, which is in line with Hawkins and Brinkhous'[72] observations that there is a large excess of this element in normal blood. Quick has demonstrated a wide margin of safety in the prothrombin factor, which may fall as low as 20 per cent without immediate danger of hemorrhage. A prothrombin concentration below 15 per cent is exceedingly serious and should be immediately combated by transfusion and other measures. These facts, it seems to us, make it perfectly clear why in these patients the loss of even small amounts of blood at operation or shortly after operation may be lethal and makes clear also, as we have previously emphasized, that preoperative transfusion cannot be expected to take care of postoperative hemorrhage. Finally, these facts put on a scientific basis Walters'[91] suggestion that a period of eight to ten hours after transfusion in some cases is the optimum and the only safe time for operation.

Hawkins'[92] experimental studies and Judd's[93] clinical observations point to anoxemia as an important factor in the hemorrhage of jaundiced patients as well as in the improvement which follows transfusion. Hawkins demonstrated an improvement in the coagulation time after the administration of oxygen, and Judd noted that in patients with hepatic

*Since this paper was completed, a negro female, with suspected malignancy of the biliary tract, revealed an initial serum volume index of 0.48. She did not respond to preparation by vitamin K and bile salts, and transfusion raised the index only to 0.70, and that temporarily. At the end of a month, in desperation, she was operated upon, with a SVI of 0.55. Exploration and biopsy revealed inoperable carcinoma of the gall bladder. She oozed freely at operation and died from intraperitoneal hemorrhage on the seventh postoperative day.

disease the oxygen saturation of the blood is markedly decreased, but can be readily improved by transfusion.

McNealy and his associates[94] advise the use of viosterol to counteract the bleeding tendency in jaundice, on the basis of an observation made by the late R. H. Jaffe and later confirmed by Greaves and Schmidt, to the effect that a deficiency of bile in the gastrointestinal tract is associated with a defective or impaired absorption of fat soluble vitamins, especially vitamin D. In the cases they studied, the administration of viosterol in 30 drop doses t.i.d., together with the administration of bile salts if the stools were acholic, practically always reduced to normal an unduly prolonged Ivy bleeding time; failure in a few cases could be explained by far-advanced or fulminating hepatic insufficiency. Boys[87] had good results with this plan of therapy except in two patients with extensive carcinomatosis. He advances the suggestion that perhaps failure under it may indicate an apparently permanent fat-soluble vitamin deficiency due to a malignant process. Johnston[95] in a similar study found the Ivy bleeding time improved in 23 of 24 cases treated by this method, and unimproved in 11 of 13 control cases. In a discussion of this report Zinniger called attention to the practical consideration that the routine prophylactic use of viosterol could scarcely be considered in public institutions, since the cost was approximately five dollars per patient. Gray and Ivy,[96] who studied the role of serum calcium fractions in the improvement following this type of therapy, found no significant alterations.

The use of vitamin K in the preparation of jaundiced patients for operation is the logical outcome of the studies which have been briefly outlined in this section. This vitamin[97] is particularly abundant in hog liver oil, cabbage, spinach, tomatoes, and alfalfa, from which a powder can be prepared, but it is not present in cod liver oil, wheat germ oil, carotene, cevitamic acid, egg albumen, or ultraviolet irradiation. Butt, Snell, and Osterberg,[98] among others, report excellent results from a preparation of fish meal, as suggested by Almquist,[75] in daily doses of 200 mg., which is almost ten times the necessary calculated dose of 23 mg. Whole human bile or bile salts were also used as indicated, to assist in the absorption of vitamin K as well as of vitamins A and D. Their studies cover a series of 18 patients with complete biliary obstruction. In view of our own unwillingness to accept liver damage as the chief cause of the hemorrhagic tendency, it is interesting to note that one patient who had demonstrated such a tendency did not exhibit it after operation, for which she had been carefully prepared, though she did lose her life from liver insufficiency. Quick, on the other hand, reported in very considerable detail a case in which the prothrombin deficiency paralleled the hepatic dysfunction, as did the improvement in both regards.

McNealy[99] has published an excellent outline of the preparation of the jaundiced patient for operation, which is complete in all details except for the very recent suggestion of the use of vitamin K and bile salts.

SURGICAL THERAPY

It is generally agreed today that exploration of the bile ducts should be routine in all cases of obstructive jaundice unless there is most absolute evidence that they are patent. The experience of all observers, of whom Lahey[100] is typical, is to the effect that as the percentage of explorations increases so does the percentage of stones found. Furthermore, since overlooked stones are the most frequent cause of secondary surgery, there is a corresponding decrease in the percentage of such operations and an actual decrease in the final mortality.

Allen and Wallace,[101] among others, advocate dilatation of the ampulla of Vater with the Bakĕs dilators. Their results are apparently good, but the recent experimental studies of Zollinger, Branch, and Bailey[102] introduce grave doubts as to the safety of the method. Their work demonstrates that the immediate result of this maneuver is hemorrhage and inflammation and that the permanent dilatation of the ampulla is actually less, as the result of scarring, than when simple cholecystectomy is done. Their clinical studies with perfusion pressures also show that the pressure is greater when the papilla is extensively dilated than when it is not explored. The plan of immediate cholangiography, as advocated by Payne[103] and by Best and his associates,[104, 105] seems equally effective and far less dangerous.

Beall[106] considers that prolonged drainage of the common duct is definitely harmful, and regrets the only two cases in which over a twenty-year period he has used the method. He notes that the single similar paper in that period, by Richter and Buchbinder, was warmly approved by Halsted. Walton[22] advocates common duct drainage only when there is definite suppuration and regards routine dilatation of the ampulla of Vater as more effective. He considers the T-tube dangerous and never employs it. Against the authorities who use common duct drainage cautiously or not at all must be set such writers as Payne[107] and Carter,[108] who advocate prolonged drainage, sometimes for several months. They determine the necessary period by studies of the function of the liver, studies of the state of the duct, and studies of the function of the sphincter of Oddi. Carter also suggests the use of specific therapy, such as bacteriophage, for the treatment of subacute pancreatitis.

Several writers, Ravdin and Frazier,[109] Culligan,[110] and Sharples,[111] have pointed out that fatal consequences may follow the strain suddenly placed upon a damaged liver by the sudden release of complete obstruction. Our own[11] experimental observations clearly establish this risk. Ravdin and Frazier have devised an ingenious apparatus for gradual

biliary decompression, which, by regulation of the bile drainage, also protects the patient from the asthenia likely to follow prolonged loss of bile salts unless these are added to his diet or he is re-fed his own bile.

According to Whipple's[112] latest contribution, the radical operation for carcinoma of the ampullary region and head of the pancreas devised by himself, Mullins, and Parsons[113] has resulted in the survival for various periods to date of 5 out of 10 patients on whom it was performed. The eleventh patient survived operation but died of cholangeitis and multiple liver abscesses at the end of five months. This is an extraordinary number of survivals in a group of conditions hitherto regarded as almost uniformly fatal.

In an experimental study of the results of various methods of exclusion of pancreatic secretion from the intestinal tract, we[114] demonstrated several facts. In the first place, the digestion of fat and protein remained approximately normal, even when unusually large amounts of these substances were included in the diet, though fatty changes invariably developed when partial or complete pancreatectomy was added to ligation and division of the pancreatic ducts. In the second place, such changes did not develop in these animals when lecithin was included in the diet, or when the ducts were merely divided and ligated and the pancreas was left in situ. These findings seem to corroborate the demonstration by Dragstedt and his co-workers[115, 116] of a pancreatic hormone which is able to take over the function hitherto assigned exclusively to the external secretion of the pancreas. On the basis of our experimental observations, it may be said that in malignancy of the ampullar and periampullar regions radical surgery which does not involve excision of the pancreatic tissue may safely be done without reimplantation of the pancreatic ducts, but that lecithin, choline, pancreatic substance, or alcoholic extracts of the pancreas must be administered to forestall fatty changes in the liver when the operation includes excision of the head or of the head and body of the pancreas.

COMPLICATIONS

Hebert[117] has continued the experimental work of Mann and Bollman[6] on the tendency of the jaundiced animal to develop peptic ulcers, particularly on the duodenal side. His studies show that in these animals the gastric acidity following test meals and the duodenal alkalinity are not materially altered, but that both the acidity and the amount of gastric secretion in the fasting stomach are decreased. There is also a reduction in the buffering capacity of the duodenal contents, which is still further reduced when ulceration is present. These findings, of course, can be applied to the human being only with many reservations, for man can live only a limited time with complete biliary obstruction, and unrelieved jaundice is rarely present for a sufficiently long time to permit comparison with experimental animals.

There is a significant and increasing emphasis in the literature on the factor of hepatic insufficiency in jaundice. Heyd,[118] who was the first to develop this association, naturally stresses it particularly, though he is careful to point out that it is merely frequent and not inevitable. Many writers now tend to emphasize the fact that hepatic dysfunction is more important than demonstrable morphologic hepatic change. It seems reasonable to believe, as Garlock[119] has postulated, that whether or not the clinical picture of hepatic insufficiency be supported by the morphologic evidence of liver damage at autopsy, there is present some functional, intracellular hepatic derangement, with associated toxic and functional renal impairment, which is fundamentally responsible for the clinical syndrome. Our personal (unpublished) studies with chloroform and carbon tetrachloride necrosis of the liver furnish some support for this point of view.

The problem of renal dysfunction in jaundice, whether per se or as a part of hepatic insufficiency, is also attracting increasing attention. Thus Meyers, Brines and Juliar,[120] in a study of 21 fatal cases of jaundice, found 7 cases with a nitrogen retention above 50 c.c. per 100 mg. of blood, though in only 2 instances was sufficient renal pathology demonstrable to account for the laboratory findings. The logical explanation in at least some of the remaining cases seemed to be that the degenerating products of liver necrosis and insufficiency were responsible for the inhibition of renal function, the lesion being so acute and therefore so brief that structural alterations had no time to manifest themselves.

Elsom[121] studied 16 patients with obstructive jaundice who presented signs of renal damage, chiefly a reduced urea clearance and an excessive excretion of casts, epithelial cells, and leucocytes, without marked hematuria and albuminuria. As the jaundice subsided, the evidences of renal injury entirely disappeared. The author points out the extreme importance of these danger signals of renal dysfunction if surgery is to be undertaken. A similar warning was issued by Wilensky and Colp[122] some years earlier, to the effect that the patient with diminished renal function is a poor risk for biliary surgery. Jones[123] repeatedly has emphasized the importance of the renal factor in hepatic disease, as well as the prognostic value of the presence or absence of spontaneous diuresis in both the jaundiced and the nonjaundiced patient.

The explanation of this type of renal function, which must be sharply distinguished from the bile nephrosis frequent in jaundiced patients, is not yet clear. In our own experimental work on the liver-kidney syndrome[11] we have been able to reproduce the deferred uremic death by the release of an artificially created biliary obstruction; the blood nitrogen values ranged as high as 171 mg. per 100 c.c. of blood. In another group of experiments[124] the intravenous and intraperitoneal injection of saline and aqueous extracts made from the livers of patients

who had died hyperpyrexic liver deaths caused elevations of nitrogen retention, which in some cases ranged to 181 mg. per 100 c.c. of blood. In the animals which recovered, the values fell to normal. Post-mortem study of both the animals which died and those which were sacrificed showed various degrees of liver changes in them all, associated with degenerative changes in the convoluted tubules of the kidneys, and mild hyperemic and avascular swelling of the glomeruli.

All these changes indicate the action of a powerful toxic agent. We[125] have shown in a series of personal experiments that the lining cells of the biliary ducts play no part in their production. Hans Meyer, quoted by Henschen,[13, 14] suggests that the liver may furnish a urine-driving hormone, and Glauback and Molitor, also quoted by Henschen, report the relief of experimental anuria by the injection of a liver-extracted fraction. It would be tempting to accept these explanations, but the evidence is far from conclusive. The most reasonable explanation is that offered by Bruhl and Watzadse and quoted by Vaccaro,[126] to the effect that a diminished glycine synthesis in the liver is the fundamental cause of the oliguria, passing over into anuria, which is characteristic of this syndrome. Such an explanation, it should be noted, would indicate that the kidney damage is not associated with jaundice per se, but with the underlying hepatic dysfunction.

MORTALITY AND PROGNOSIS

It is generally granted that the mortality of jaundiced patients is considerably higher than that of nonjaundiced patients, but otherwise there is no very general agreement about results. Lee and Totten[127] consider the prognosis of common duct malignancy more favorable than the prognosis of other malignancies of the gastrointestinal tract because of the early appearance of jaundice. But their qualification, "except for the complications and duration of the jaundice," partially invalidates the rather optimistic outlook. Furthermore, as Lampert[128] pointed out in a discussion of carcinoma of the hepatic duct, to which the same guarded optimism is sometimes applied, malignancy of the biliary ducts often presents insuperable technical problems, quite aside from the fact that in cancer even early symptoms are late symptoms.

Best and Hicken,[129] in a discussion of the abnormally high death rate for short-circuiting operations in obstructive jaundice, conclude that an important cause is failure to determine the patency of the common duct before the type of surgery is decided upon. If its patency cannot be established by the probe or by cholangiography at the operating table, choledochostomy or choledochoduodenostomy, although technically more difficult, will give better final results and a lower mortality than cholecystostomy or gall-bladder intestinal anastomosis.

The average duration of life after a short-circuiting operation for malignancy is not more than seven or eight months, according to the

statistics of Clute,[130] of Eliason and Johnson,[131] and of Rives, Romano, and Sandifer. Oppenheimer[132] reports the survival of a patient with carcinoma of the head of the pancreas for twenty-nine months after cholecystgastrostomy, but this is most unusual; autopsy confirmed the diagnosis and revealed no metastases and no ascending infection, the usual causes of death in patients who survive the actual surgical act. Clute points out that, while exploration should never be omitted, the mere opening of the abdomen is attended with a very high mortality. Eliason and Johnson conclude that a normal life expectancy is possible in nonmalignant disease, but warn that the mortality from ascending infection is sufficiently high to make the indiscriminate performance of short-circuiting operations entirely unjustifiable.

Surgery for operative injuries of the common duct is always difficult and is attended with a rather high mortality, but the results may be excellent, as Lahey, Eliot, and others have shown. The mortality of surgery for uncomplicated stone in the common duct, although somewhat higher than for simple cholecystectomy, has been reduced to incredibly low figures in the hands of expert surgeons.

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Review of Recent Meetings

REVIEW OF THE TWENTY-FIRST ANNUAL MEETING OF THE AMERICAN SOCIETY FOR THORACIC SURGERY, ATLANTA, GA., APRIL 4-6, 1938

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(From the Department of Surgery, Medical College of Virginia)

Dr. Stuart Harrington, Rochester, Minn., gave the presidential address on **Hiatus Hernia of the Diaphragm.** He showed some excellent anatomic sections made with especial elastic tissue stains, illustrating the weakening of the elastic fibers in the gastrophrenic ligament in patients with hernia. He gave a number of reasons for his preference for the abdominal approach; among these were: (1) there was less chance of postoperative pulmonary complications; (2) it was much easier to expose and repair a hernia projecting through the diaphragm on both sides of the esophagus or projecting through posterior to the lower end of the esophagus; and (3) the attachment of the hernial sac to the stomach could be separated only when the operation was carried out through this approach. In 21 cases in which the major operation did not seem advisable, phrenic interruption was done; only 5 of these patients failed to receive any relief. He showed an excellent motion picture of the technique of the transabdominal operation.

Dr. Joseph Weinberg, of Omaha, Neb., read a paper on the treatment of acute empyema thoracis by open intercostal drainage. He reported 53 consecutive cases operated upon by this method without a death. The operation is carried out in adults under local anesthesia and in children under cyclopropane. The intercostal muscles, the pleura, and the muscles of the chest wall overlying the intercostal space, usually the eighth posterolaterally, are excised for a distance of 5 to 8 cm. The opening is temporarily blocked with a piece of rubber dam packed with gauze, so as to permit the escape of pus, but at the same time to prevent the entrance of air into the pleural cavity. This tampon is removed at the end of forty-eight hours and irrigations are begun. He avoids the use of small pieces of gauze and short tubes for fear they will be lost in the cavity. He feels that irritation of the lungs by stiff rubber tubes may be one of the causes of the spread of infection by infected emboli. In patients under 16 years of age, the average postoperative convalescence period was twenty-one days, and in those over 16 years it was fifty days. He emphasized the importance of waiting until the fluid becomes definitely purulent before establishing this type of drainage.

Dr. I. A. Bigger, Richmond, Va., reported 17 patients with heart wounds operated upon in the Medical College of Virginia Hospitals during the past eight years and discussed the treatment and prognosis of heart wounds in general. He

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suggested that these patients be divided into four groups, depending upon whether the hemorrhage into the pleural cavity or tamponade predominated in the picture and also upon the degree of hemorrhage and the degree of tamponade. He advised immediate operation when there was massive hemorrhage into the pleural cavity and when there was tamponade with persistent circulatory collapse. Conservative treatment was advised in those patients with only moderate hemorrhage into the pleural cavity and aspiration of the pericardium in those with tamponade, but with a rise in the systolic blood pressure to near normal after the administration of fluids, adrenalin, etc.

He compared the recovery rates as estimated by the collection of cases from the literature with those collected by a questionnaire sent to the members of the American Association for Thoracic Surgery, the American Surgical Association, and the Southern Surgical Association. The latter figures showed a recovery rate of approximately 50 per cent, which was presumably a more accurate index of the chances of recovery from a heart wound than the figures obtained from the current literature.

In discussing this paper, **Dr. Alfred Blalock,** Nashville, Tenn., emphasized the importance of venous pressure determinations in the diagnosis of the condition. He also advised against prolonged delay before operation because of the danger of circulatory collapse. **Dr. D. C. Elkin,** Atlanta, Ga., doubted the wisdom of conservative treatment in patients with heart wounds, even when the blood pressure and pulse returned to a relatively normal level following conservative treatment, and reported one patient in whom conservative treatment was carried out. The patient at first had signs of a moderate tamponade which gradually improved, but, on about the sixteenth day after admission, the child developed signs of severe tamponade and died, apparently from secondary hemorrhage into the pericardium.

Dr. W. E. Adams and (by invitation) **Dr. Dallas B. Phemister,** Chicago, Ill., made a report on some experimental work on resection of the lower end of the esophagus and anastomosis with the stomach above the diaphragm. Sixty-one dogs were used and in 32 of them an anastomosis of the stomach with the esophagus was performed after resection of varying lengths of the lower esophagus. In 13 of these the anastomosis was made after the stomach had been brought up through a new opening in the diaphragm and in 19 the stomach was brought above the diaphragm through an enlarged hiatus. In 2 instances a continuous suture was used for the anastomosis and both dogs developed stenosis at the suture line. In 15 dogs the entire esophagus was mobilized and brought out through a cervical incision. All of these died in from two to eight days. In 14 dogs short segments of the thoracic esophagus were resected and the dogs were fed through a gastrostomy tube. Only very high resections were tolerated.

They reported a successful resection of the lower end of the esophagus in a 53-year-old woman with a carcinoma of the cardiac end of the esophagus. A transthoracic approach was used; the cardiac end of the stomach and the lower end of the esophagus were resected, the opening in the stomach closed, and the lower end of the esophagus anastomosed with the fundus of the stomach above the diaphragm. The left thoracic cavity was drained by one catheter. The patient made a relatively uneventful recovery.

Dr. Carl Eggers, New York, N. Y., read a paper on **Upper Esophagostomy: Its Indications and Uses.** He stated that gastrostomy should always be done as a preliminary procedure. At the time of the gastrostomy he advises dividing the

upper end of the esophagus and implanting the distal end of the upper segment beneath the skin near the left sternoclavicular junction. The upper end of the distal segment is closed by inversion and dropped back into the thorax and the resection carried out at a later stage.

In discussing Dr. Adams' and Dr. Eggers' papers, **Dr. Edward D. Churchill,** Boston, Mass., **Dr. W. D. Andrus,** New York, N. Y., and **Dr. Harold Brunn,** San Francisco, Calif., also advised doing a laparotomy first in lesions near the cardia to be sure that there were no metastases. **Dr. Richard Overholt,** Boston, Mass., advised against gastrostomy in the lower esophageal lesions because of fixation of the stomach resulting from this operation. **Dr. Franz Torek,** Montclair, N. J., stated that he did not believe that esophagogastrostomy was feasible in most esophageal tumors because they are located too high within the thorax. Dr. Adams stated that a satisfactory exposure and exploration of the abdomen could be had through the diaphragmatic incision.

Dr. Evarts Graham and (by invitation) **Dr. Brian Blades,** St. Louis, Mo., reported upon 52 mediastinal tumors studied in the Washington University Clinic. In only 23 was operation attempted. Forty per cent of the entire group were of lymphatic origin; 11.5 per cent arose from nerve tissue; and 23 per cent were teratomas or dermoids. About 4 per cent were sarcomas from other than lymphatic tissue. Five of the teratomas were malignant and inoperable. A sixth one recurred after operation. Six were benign. Dr. Graham emphasized the importance of operating upon teratomas and dermoids early because of the danger of the development of malignancy or infection. Operation is frequently delayed because both physicians and laymen believe that the mortality rate in operation on such tumors is extremely high. He stated that the mortality rate in operation for benign tumors is approximately 7 per cent, while the chances of the development of malignancy are about 66 per cent. If there is doubt as to the diagnosis or the type of tumor, roentgen ray therapy should be tried. If the tumor is of lymphatic origin, it will shrink rapidly following such treatment; whereas, teratomas and dermoids do not show an appreciable response.

He advised intratracheal cyclopropane anesthesia and in most instances approaches the tumors through an anterior intercostal incision. In female patients this incision is made below the breast and when the operation is carried out in this way without the removal of cartilages the scarring is practically imperceptible. When the tumor is definitely in the posterior mediastinum, a posterior approach is used.

Dr. Blades stated that the anterior approach was followed by very slight pain and the convalescence was short (three to four weeks). He advised aspirating the pleural cavity on the first and second postoperative days.

Dr. John C. Jones, Los Angeles, Calif., and **Dr. Harold Brunn,** San Francisco, Calif., reported substernal thyroids which had been mistaken for dermoids. **Dr. Edward D. Churchill,** Boston, Mass., stated that the fear of chest deformity following thoracic operations was the result of the large number of chronic empyemas operated upon following the War period.

Dr. J. Samuel Binkley, New York, N. Y. (by invitation), discussed **Aspiration Biopsy of the Lung** as done at the Memorial Hospital in New York. The aspiration is done with the patient in the upright position. An 18 caliber needle is used and the material obtained is immediately smeared on a slide and stained. Ninety-two aspiration biopsies of the thorax have been done in this hospital during the

past ten years. In 56 cases of bronchogenic carcinoma there were 60 per cent positive biopsies by aspiration, while there were 57.1 per cent bronchoscopic failures as proved by aspiration biopsies.

In discussing Dr. Binkley's paper, **Dr. J. J. Singer,** Los Angeles, Calif., and **Dr. Ralph B. Bettman,** Chicago, Ill., warned of the dangers of pneumothorax, empyema, and embolism. Dr. Singer advocated artificial pneumothorax followed by thoracoscopic examination and biopsy. **Dr. D. C. Elkin,** Atlanta, Ga., stressed the importance of a positive diagnosis in suspected carcinoma of the lung, for otherwise the patients would not submit to operation.

Dr. William M. Tuttle and (by invitation) **Dr. J. P. O'Connor,** Detroit, Mich., discussed extrapleural thoracoplasty in the presence of contralateral pneumothorax. Eighty-six patients who had pneumothorax on one side were subjected to thoracoplasty on the contralateral side. Fifty-one of these showed cavitation; in 18 the cavities remained open, but in 69 per cent they were closed following thoracoplasty. Twenty-seven of these patients still have a pneumothorax. The mortality rate was 21.1 per cent; 9.4 per cent died shortly after operation and in 11.7 per cent death occurred late. Twenty-two per cent of the patients showed arrest of the disease.

Dr. Pol N. Coryllos and **Dr. G. G. Ornstein,** New York, N. Y., discussed **Tension (Giant) Tuberculous Cavities; Pathogenesis, Mechanics, and Surgical Management.** Dr. Coryllos stated that these large round cavities contain air under positive pressure with a higher percentage of oxygen and less carbon dioxide than alveolar air. He showed diagrams illustrating the effects produced by varying degrees of bronchial stenosis. In moderate stenosis the air passes both ways relatively freely. In more advanced stenosis air is able to enter the cavity on inspiration, but part of it is trapped on expiration, thus producing a constant positive pressure within the cavity, thereby causing it to enlarge rapidly. In complete stenosis atelectasis results. These authors have demonstrated by gas analysis and by the injection of lipiodol directly into the cavity through the chest wall that there is a communication with the bronchial tree in these cases. The trapping of air in the cavity and the production of a positive pressure produce a vicious cycle, and reduction of the pressure within the cavity by aspiration of air may control this vicious cycle and allow air to escape on expiration.

Dr. Coryllos believes that the only cure for such cavities is obliteration of the bronchial opening by the application of sclerosing substances or by muscle transplants. He stated that the experiments so far have not been productive of positive results but have opened up an interesting problem for further investigation.

Dr. Jerome R. Head, Chicago, Ill., described a multiple stage muscle-splitting operation for extrapleural thoracoplasty which he had done first in 1928 but had then abandoned. More recently he had again begun doing this type of procedure because it reduces shock and infection to a minimum and decreases paradoxical respiration. It also preserves the muscles of the shoulder girdle. It is carried out in six stages, three posteriorly and three anteriorly. Most of the rib stripping is done subcutaneously by an especially constructed periosteotome. The periosteum of the upper six ribs posteriorly is destroyed. Apicolysis may be done in conjunction with the upper posterior stage.

Dr. Owen H. Wangensteen, Minneapolis, Minn., also discussed a method for doing thoracoplasty through short incisions. The operation is done through short anterior and posterior incisions, which depended initially upon the direction and insertion of the external intercostal muscle bundles. The lower borders

of the ribs may be stripped through anterior incisions as far back as the rib angles. Short segments are removed anteriorly and about two weeks later through a 10 cm. posterior incision the superior rib borders are stripped and the ribs removed. At the same time the ribs below those removed are divided posteriorly so that they can be removed at the third stage through an anterior incision. He has found it possible by this method to obliterate total empyema cavities completely by a three-stage operation. In some of the very large empyema cavities there has been a small residual cavity posteriorly necessitating excision of parietal pleura.

In his more recent technique the greater portion of the operation is carried out from in front. With development and employment of sharp periosteal separators, the rib may be removed from either direction, granted that it is divided on the other end. If a rib is divided near the sternum, it may be completely removed through a posterior incision; if a paravertebral segment of the rib as far as its angle be removed through a posterior incision, the remainder of the rib may then be removed with ease through an anterior incision. This method of rib removal is particularly applicable in the performance of thoracoplasty for tuberculosis as well as for chronic empyema. If apicolysis is to be done at the time of thoracoplasty for tuberculosis, it is carried out from in front, the apex of the lung is stripped downward as far as is indicated and fastened down by broad bands of catgut, such as are used by urologists to suspend kidneys.

Dr. Joseph W. Gale and (by invitation) **Dr. W. H. Oatway, Jr.,** Madison, Wis., discussed the results and complications of the Semb type of apicolysis carried out in conjunction with thoracoplasty. Dr. Gale removes from two to four ribs at one time and does an apicolysis down to the upper intact rib. He then sutures the posterior ends of the intercostal muscles to the neck of the first intact rib posteriorly to keep the apex of the lung down. He uses cyclopropane anesthesia and states that a relatively deep anesthesia is necessary for the operation.

This operation has been carried out in the University of Wisconsin Clinic in 102 cases over a two-year period. Thirteen of these patients were more than 45 years of age. There was some involvement in the contralateral lung in 84 per cent of them and there were advanced lesions in the contralateral lung in 24 per cent. He suggests that both the pre- and postoperative administration of opium be reduced to a minimum and that oxygen therapy be continued as long as there is evidence of anoxemia. The chief complication has been atelectasis which frequently comes on after apicolysis. He advises clearing the bronchial tree in wet cases. Atelectasis may be treated by bronchoscopic aspiration.

An effective collapse was obtained in 77 per cent of the cases. There were 14 deaths in the group, or a mortality rate of 13.7 per cent. He believes that the ideal cases for the Semb operation are those patients in good condition with a good contralateral lung, in whom other systemic complications are absent and where the cavity is large, thin-walled, and easily collapsible. The use of apicolysis in an unselected group allowed the authors to derive the first tentative indications for the operation. They conclude that in repeating the same cases they would decrease the extent of lysis in 40 per cent of the cases and not do a lysis in another 20 per cent.

Dr. Ethan Flagg Butler, Ithaca, N. Y., described **Myoplastic Thoracoplasty.** The operation is indicated especially in large anterior apical cavities and is carried out through an anterolateral incision, extending from the midclavicle to the anterior axillary line at the level of the fourth interspace, and thence downward an additional three inches. The insertions of both pectoral muscles are

divided. The first rib may be left in place, but the second and third ribs are removed from sternum to vertebral bodies. The apex of the lung is stripped down to the desired level and the pectoral muscles are swung across above the lung and sutured to the posterior wall so as to form a muscle diaphragm for holding the lung down.

The operation has been carried out in 10 cases with 2 deaths, one from a spontaneous pneumothorax in the other lung and the second from the late effects of wound infection attributable to a pre-existing mixed infection empyema. Of the 10 cases, 3 have shown entirely satisfactory results, 1 is questionable, and 4 have been unsatisfactory. Dr. Butler now thinks that it is probably better to remove the first rib to avoid the formation of a dead space.

In the discussion of this group of papers on pulmonary tuberculosis, **Dr. Edward S. Welles,** Saranac, N. Y., raised the question as to whether there might be lymphatic blockage of the breast following Dr. Butler's operation. He reminded the audience that Dr. Archibald had used a muscle flap in his apicolysis but had given it up because of the marked degree of muscle atrophy which followed. He advised against using the Semb operation as a routine procedure and stressed the importance of better x-ray plates, particularly the importance of overexposure of the involved side in an attempt to demonstrate residual cavities in a collapsed lung.

Dr. Cameron Haight, Ann Arbor, Mich., stated that they were no longer using the Alexander type of muscle transplant to the apex. He also said that they had given up cavity drainage in large tuberculous cavities. **Dr. J. J. Singer,** Los Angeles, Calif., called attention to bronchiectasis in association with pulmonary tuberculosis as being one of the causes of failure of thoracoplasty. **Dr. Warriner Woodruff,** Saranac, N. Y., also called attention to the dangers of the Semb operation and warned against its too frequent use. He had found that it increased the occurrence of atelectasis and also increased the chances of tuberculous infection of thoracoplasty wounds, probably due to the division of infected mediastinal lymph channels during the dissection. **Dr. Richard Overholt,** Boston, Mass., expressed disappointment at the results obtained by apicolysis but stated that he felt that it was of some value. He still favors the large posterior incision because he feels that shock is not due to the amount of muscle and skin severed but primarily to the amount of rib removed, and to control this he advises waiting a sufficient time between stages. **Dr. Pol N. Coryllos,** New York, N. Y., pointed out the trend toward complete selective collapse, especially in bilateral cases. He stated that paralysis of the diaphragm increased the chances of atelectasis, a condition which should always be kept in mind and treated by aspiration when it occurred. **Dr. Joseph Gale,** Madison, Wis., emphasized the importance of postoperative compression of the chest by the John Steel type of brace. Dr. Butler advised doing wet cases in the afternoon, to give them a chance to clear the cavities out during the morning.

Dr. Ronald Belsey, London, England (by invitation), introduced by **Mr. J. E. H. Roberts,** London, England, and **Dr. Edward D. Churchill,** Boston, Mass., discussed **Extrapleural Pneumothorax,** a procedure introduced by Graf and first tried by Roberts in 1935. It apparently is indicated in patients in whom thoracoplasty would be desirable but for certain reasons is contraindicated. The operation is carried out under cyclopropane anesthesia and an incision is made over the fourth rib centering over its angle. The rib is resected posteriorly as far as the transverse process; the pleura is then exposed and separated from the chest wall by careful dissection until a sufficient amount is free to give the desired collapse.

Careful hemostasis is required and it is, of course, necessary to make an airtight closure. Frequent refills are necessary in the early stages, two a day for the first few days, but eventually only one refill every two or three weeks will be needed. It is necessary at first to maintain a positive pressure of 10 to 20 cm. of water.

Serosanguineous effusion occurred in practically all cases. This should be aspirated. In one instance infection developed in this fluid and the cavity had to be drained. One patient died from hemorrhage. The temporary results were satisfactory in most instances. It is not to be used when thoracoplasty is suitable. The pleura is usually stripped down to the level of the seventh or eighth rib posteriorly. The most common complications are serosanguineous effusion, injury to the pleura, emphysema, and hemorrhage. Infection is apt to be serious.

This paper was discussed by **Dr. Churchill,** who stated that he felt that the procedure was of value, but that he wished to emphasize that it was more dangerous than thoracoplasty. **Dr. John C. Jones,** Los Angeles, Calif., emphasized the absence of deformity following extrapleural pneumothorax in young children. **Dr. Coryllos** insisted that the operation should be called pleurolysis and said that it was not a satisfactory procedure where there was fibrosis. He felt that it was a suitable procedure for cases with bilateral disease where thoracoplasty was contraindicated. He does the operation under local anesthesia and uses a Cameron light for better vision. He uses small sponges soaked in novocain and adrenalin with which to strip the pleura.

Dr. John C. Jones, Los Angeles, Calif., discussed the indications and contraindications for lobectomy and pneumonectomy in pulmonary tuberculosis. He reported four cases in which such procedures had been carried out. The first one, a man, had the right upper and middle lobes removed for uncontrollable hemorrhage. Thoracoplasty was done later for obliteration of the cavity. The second patient, a 28-year-old woman, had stenosis of the left main bronchus with severe bronchiectasis distal to it, for which pneumonectomy was done. There was a persistent bronchial fistula. Eight weeks after the pneumonectomy she developed an exudative lesion in the right lung, but this is now apparently improving. The third case, a 29-year-old female with a large apical cavity, had had an extensive thoracoplasty in 1936, but without obliteration of the cavity. There was no evidence of tracheobronchial tuberculosis. The upper lobe was removed and there was demonstrated a 2½ cm. residual tuberculous cavity. She developed a bronchial fistula which has now practically healed over. The fourth case, a 49-year-old woman, had a lesion of the right lower lobe which was thought to be malignant. Pneumonectomy was done through the anterior approach and there was a relatively uneventful convalescence.

The indications for lobectomy or pneumonectomy are: (1) failure to respond to other procedures; (2) persistent hemorrhage; (3) suppurative lung disease and tuberculosis in the presence or absence of bronchial occlusion; (4) persistent cavitation after complete thoracoplasty; (5) atelectatic honeycombed lobe after complete thoracoplasty; and (6) basal cavities which fail to heal after the usual procedures.

In discussing this paper, **Dr. Evarts Graham,** St. Louis, Mo., recalled that Tuffier removed a portion of the right lung and found it to be tuberculous approximately fifty years ago. He (Dr. Graham) had done two lobectomies to arrest hemorrhage and three others for calcified lesions which were in all probability tuberculous in origin and one other lobectomy for what was thought to be a tumor but which proved to be a tuberculoma. He emphasized the impor-

tance of not attempting to develop this phase of tuberculosis surgery too rapidly. **Dr. Robert Glen Urquhart,** Norwich, Conn., felt that tuberculous stenosis of the bronchus was a sound indication for pneumonectomy.

Dr. H. McLeod Riggins, New York, N. Y., discussed **Tuberculosis of the Bronchi Complicating Pulmonary Tuberculosis: Its Effect Upon Closure of Pulmonary Cavity.** He stated that tuberculous endobronchitis secondary to pulmonary tuberculosis was a serious complication, making the patient a poor surgical risk and increasing the danger of emphysema and atelectasis. He classified these lesions as: (1) catarrhal, (2) ulcerative, (3) hyperplastic, and (4) fibrostenotic. The bronchial lesion interferes with the ability of the lung to eliminate secretions and also increases the pressure within the lung, thereby causing an increase in the size of cavities. He stated that the bacterial flora within the bronchi altered the clinical picture. The recommended treatments have been: (1) postural drainage, which is of questionable value; (2) pneumothorax, which is distinctly dangerous; (3) bronchoscopic treatments, which are valuable especially when used in conjunction with silver nitrate, the electric cautery, and ionization. In his group of patients, 30 per cent were worse, 45 per cent were improved, 8 per cent healed, 4 per cent unknown, and 11.7 per cent dead.

Dr. J. J. Singer, Los Angeles, Calif., in discussing this paper, stated that many patients who were repeatedly bronchoscoped felt better afterwards, but after a time this was dependent not so much upon the drainage as upon the repeated use of cocain. He felt that bronchoscopic drainage was palliative at best and urged earlier resort to surgery.

Dr. Oscar Auerbach, New York, N. Y., read a paper on **Bronchopleural Fistulas Complicating Pulmonary Tuberculosis: A Clinical Pathological Study.** He stated that it was the commonest cause of infection of the pleura. Out of 1,000 cases of fibroulcerative tuberculosis, 78 patients developed bronchopleural fistulas; 67 of them had single fistulas, and 11 had multiple fistulas. He classified them as: (1) fistulas without pre-existing pneumothorax, which are usually fatal; (2) fistulas complicating artificial pneumothorax; many of these patients survive and they account for 70 per cent of his series; (3) fistulas developing after internal pneumolysis operations (tuberculous caseation, excavation, on to fistula formation). The second group occurs from two to six months to four years after the institution of pneumothorax; they are caused by the liquefaction of small caseous foci or cavities communicating with a bronchus and breaking through into the pleura. They are apt to follow collapse of acute exudative lesions, so this may be considered a serious contraindication to collapse in this type of case, as the collapse seems to increase the chances of certain areas' sloughing out, thus leading to fistula formation. The third group (after the Jacobeus operation) accounted for 1.25 per cent of Dr. Coryllos' series.

Adhesions which may be cut are of three types: (1) fibrous (hyalinized connective tissue), which are safe to cut; (2) round bands, often containing lung tissue; and (3) fanlike bands, always containing lung tissue. When lung tissue is cut, necrosis ensues and empyema develops.

Healing in fistulas may occur spontaneously, due to obliteration by thick pleura. The pleural layers may stick together or exudate may seal the fistula and form a fibrous scar. The temporary closure often occurs due to fibrinous plugs. The size of the fistulas varies from 1.0 mm. to 4.0 cm. Sixty per cent are in the upper lobes.

In discussion **Dr. Coryllos** said that bronchopleural fistula was rare without pneumothorax because of the protection afforded by pleural adhesions. All

fistulas lead to empyema. There are no symptoms if the fistula is small, and it can be detected only by gas analysis. He accounted for his low rate of fistulas with the Jacobeus operation by stating that he worked only in the parietal subpleural space.

Dr. Alton Ochsner and (by invitation) **Dr. Michael DeBakey,** New Orleans, La., described **The Operative Correction of Pectus Excavatum.** It is usually congenital, consisting of depression of the sternum and costal cartilages, and is not due to rickets. It occurs four times as frequently in men as in women. The various causes to which it is attributed are that it runs in families, is due to growth disturbances, perhaps is due to pituitary influence, is due to mediastinitis, with resulting inward pull on the sternum. Few cases are due to acute trauma, occupation (as cobbler's disease), or poor posture.

It is characterized by depression and rotation of the sternum, a decrease in the anteroposterior thoracic diameter, and an increase in the transverse diameter. The heart is displaced usually to the left, with rotation and compression. The lungs may be compressed, as may the esophagus; and the diaphragm is depressed. Signs and symptoms may be absent. If present, they consist of: (1) cardiac: dyspnea, palpitation, pain, and decompensation; (2) pulmonary: dyspnea, cyanosis, cough, reduction in vital capacity; also dysphagia and dyspepsia; the patients are weak, asthenic, and debilitated. It is to be differentiated from rickets. Complications include myocarditis, bronchitis, and tuberculosis.

The treatment may be conservative, such as breathing, posture, and orthopedic exercises. Three types of operation have been used: (1) chondrosternal resection (10 cases with 2 deaths); (2) T-sternotomy with splitting of the sternum and some sort of wedging apart of the sternal segments (14 cases done, with 8 cures, 4 deaths, and 2 failures); (3) sternal mobilization with chondral resection (8 cases done, with 7 cures and 1 failure). The operation is done only for marked symptoms and disability. Dr. Ochsner reported a case in which he had done the latter type of operation through elliptical incisions, freed and elevated the sternum over a Parham band, which he attached to an anterior plaster shell. The result was quite satisfactory.

In discussion, **Dr. Jerome Head,** Chicago, Ill., reported two cases operated upon, with one death, and said that symptoms may be present even if the heart can escape to the left. **Dr. James C. Sandison,** Atlanta, Ga., reported two cases, one in which he used a sternal bone (tibial) wedge, and the other, a Parham band, which he advised not putting too high. Dr. Ochsner said that he would use two Parham bands instead of one on his next case.

Dr. John V. Bohrer and (by invitation) **Dr. Charles W. Lester,** New York, N. Y., discussed **Late Results of Lobectomy for Bronchiectasis in Children.** They made a careful survey of the 4 cases reported before the American Association for Thoracic Surgery in 1934 and added 6 cases operated upon since that time. Eight of the 10 cases recovered and are now in good general health. The authors pointed out the fact that these patients had been operated upon during the past nine years and for this reason the mortality rate was higher than would be likely at present because of the important recent improvements in the technique of the operation. One patient was subjected to a single stage pneumonectomy; 2 had complete pneumonectomies done in two stages (the lower lobe being removed first); the right middle, and lower lobes were removed in 1 case; and 6 had single lobes removed.

Where all of the diseased tissue was removed, cure has been complete. In those patients with slight involvement of the upper lobe at the time of removal

of the lower lobe, the disease has progressed, necessitating removal of the upper lobe. Even following complete pneumonectomy there has not been any appreciable deformity. The authors conclude that extirpation of the diseased lung tissue is the only means of curing bronchiectasis.

Dr. Edward D. Churchill, Boston, Mass., said that lobectomy really cures children even if they do not appear sick, and many bronchiectatic children on sound regimes do not look sick. The disease must be proved by lipiodol. It progresses very slowly. Fibrosis and emphysema occur in the good portions of the lung from constant infection.

Dr. Minas Joannides, Chicago, Ill., spoke on the **Mechanics of Pulmonary Abscess and Bronchiectasis with Suggested Method of Treatment.** He stated that abscess may follow pneumonia, trauma, aspiration, embolus (which causes infarct); and the organisms are fusiform bacilli, spirochetes, and diphtheroids. Infection is facilitated when blood is present in the bronchial tree. The course is broadly the same as infection and abscess formation in other tissues. Hyperemia, stasis, exudation go on; the abscess forms under the condition of low oxygen tension. The treatment is oral hygiene, postural drainage, x-ray and bacterial study, arsphenamine, and increased moisture in the air in the room, including gomenol in the steam. This causes hyperemia, increase in sputum, and increased coughing and removal of exudate. (This may be overdone and may produce pulmonary edema.)

Dr. Joannides stressed the importance of early differentiation of ordinary pneumonia and the pneumonitis which precedes abscess. He said that fusospirochetes should always be looked for in the sputum. Proper treatment in this stage will likely abort the infection. Neo- or sulpharsphenamine in 0.3 to 0.6 gm. every two or three days is a great aid. Artificial pneumothorax is definitely contraindicated, as it promotes atelectasis and produces a spread of the infection.

In discussion, **Dr. Joseph Weinberg,** Omaha, Neb., said that the mechanism of infection was much more important than the organisms, that treatment varied with the organisms, and that iodized oils should not be used. **Dr. Jerome R. Head,** Chicago, Ill., said that fusospirochetes were seen only in poor patients; such patients get intoxicated (to the point of anesthesia) and aspirate from dirty teeth and gums. Ninety per cent of these abscesses are located posterior to and below the spinal angle of the scapula. **Dr. Ralph B. Bettman,** Chicago, Ill., said that gentian violet put into the mouths of anesthetized dogs was always found in the lungs when the dogs were sacrificed.

Dr. Otto C. Pickhardt, Dr. William H. Stewart, and **Dr. Grant Thorburn,** New York, N. Y., showed some interesting moving pictures, **Cinefluoroscopic Studies of Peculiar Breathing and Chest Motion,** after phrenicotomy, thoracoplasty, pneumothorax, etc. This technique appears to have great possibilities for teaching.

I wish to express my appreciation to Dr. F. C. Fishback, Washington, D. C., for the use of his notes on the meeting in completing this report.

REVIEW OF THE THIRTIETH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CLINICAL INVESTIGATION, ATLANTIC CITY, N. J., MAY 2, 1938

NORMAN E. FREEMAN, M.D., PHILADELPHIA, PA.

TWENTY-FIVE papers were presented. The following subjects were considered to be of particular interest to surgeons:

Frederick J. Pohle and **F. H. L. Taylor,** of Boston, Mass., discussed the coagulation defect in hemophilia and described the use of a globulin substance derived from beef plasma as a local hemostatic agent in this condition. Excellent results were obtained.

William Dameshek and **Steven O. Schwartz,** of Boston, Mass., were able to produce hemolytic anemia in the experimental animal by the injection of hemolytic sera. The hemolytic syndromes were characterized by spherocytosis, increased fragility, and reticulocytosis.

Edwin E. Osgood, of Portland, Ore., studied the comparative effects of sulfanilamide and antipneumococcus serum on the growth of pneumococci obtained from human bone marrow. Both serum and sulfanilamide inhibited the growth of the organisms, but the greatest effect was observed when they were used together. From this observation it was suggested that pneumococcus infections might well be treated by the simultaneous administration of specific serum and sulfanilamide.

W. T. Salter and **F. N. Craig,** of Boston, Mass., found that the plasma of hyperthyroid patients conferred an increased metabolism upon tissue slices obtained from pedigreed mice. The increase in oxygen utilization of the surviving tissue after incubation in plasma was proportional to the increase in basal metabolic rate of the patient. The plasma of normal subjects or of subjects in whom the basal metabolic rate was increased because of leucemia did not produce any increase in the oxygen consumption of tissue slices.

Allan T. Kenyon, Irene Sandiford, A. Hughes Bryan, Kathryn Knowlton, and **F. C. Koch,** of Chicago, Ill., administered testosterone proportionate to eunuchoids and to one eunuchoid of pituitary origin over prolonged periods of time. They observed marked masculinization of the genitals, prostate, and secondary sex characteristics. There was no effect on spermatogenesis. The beneficial effect persisted only as long as medication was continued.

H. B. Friedgood and **S. L. Gargill,** of Boston, Mass., studied the androgen excretion in the urine of two patients with virilism from adrenal cortical tumors. In both patients the androgen determinations by chemical analysis exceeded 300 international units. After complete removal of the tumor in one patient, the androgen values fell to normal. In the other patient complete removal of the tumor was not possible. The increased androgen excretion persisted. In eight cases of hirsutism in which no adrenal cortical tumor could be demonstrated, the androgen excretions were below 300 international units.

Tom D. Spies and **William B. Bean,** of Cincinnati, Ohio, found that nicotinic acid in addition to preventing pellagra was also effective in preventing roentgen sickness. An increased porphyrinuria was observed in the latter condition.

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John Russell Smith and **William Bryan Kountz,** of St. Louis, Mo., perfused cadavers immediately after death with oxygenated, defibrinated blood. With a constant arterial injection of 60 c.c. per minute, the blood pressure of normal subjects was found to be lower than the pressure observed in subjects which had exhibited arteriosclerosis or hypertension before death. Injection of pressor substances produced a marked increase in blood pressure in the hypertensive subjects, but in the arteriosclerotic group the rise was less than in the normals.

Eugene A. Stead, Jr., and **Paul Kunkel,** of Boston, Mass., described a plethysmographic method for the determination of the blood flow through the foot. At a temperature of 43° C., the average flow of blood was found to be 17 c.c. per minute per 100 c.c. foot volume, only one-half as great as that through the hand at the same temperature. In patients with asymptomatic obliterative vascular disease, the flow was reduced by 50 per cent. In patients with symptoms of vascular insufficiency the flow was reduced by more than 60 per cent. Vasomotor reactions were observed in the blood flow through the foot comparable to those observed in studies on the hand.

George P. Robb and **Israel Steinberg,** of New York, N. Y., detailed a practical and safe method of visualization of the chambers of the heart, the pulmonary circulation and the aorta in man. They injected 70 per cent diodrast into the arm vein with great rapidity and, by x-ray studies taken 1 to 2 seconds later, were able to outline the vena cava, right auricle, right ventricle, and pulmonary arteries. Eight to sixteen seconds later (the circulation time was measured by the cyanide method), they were able to outline the left ventricle and aorta. In over 100 injections, no serious mishaps were observed, although a fall in blood pressure and rise in heart rate were frequent, while chills or urticaria were observed in about 10 per cent of the cases.

Jerome W. Conn and **L. H. Newburgh,** of Ann Arbor, Mich., studied the carbohydrate metabolism in six cases of chronic infectious hepatitis. The characteristic findings in all the cases were a low fasting blood sugar and an inability to store carbohydrate. When deprived of carbohydrate in the diet for forty-eight hours, three of the patients went into hypoglycemic shock. All patients showed a diabetic type of sugar tolerance curve. The carbohydrate utilization, however, was unaffected. The essential feature appeared to be liver damage with consequent inability to store or release glycogen. Removal of a diseased gall bladder brought about complete return to normal function in two of the patients.

THE FIFTY-FOURTH MEETING OF THE AMERICAN ASSOCIATION OF ANATOMISTS, UNIVERSITY OF PITTSBURGH, PA., APRIL 14-16, 1938

EDWARD A. BOYDEN, PH.D., MINNEAPOLIS, MINN.
(From the University of Minnesota)

THIS meeting, held in conjunction with the American Association of Physical Anthropologists, was of special interest as marking the fiftieth anniversary of the founding of the Association, in recognition of which a limited issue of medallions of the first president, Joseph Leidy, of Philadelphia, had been modeled by the late Dr. Robert Tait McKenzie, distinguished American sculptor and former member of the Association.

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At the subscription dinner, further commemorating this anniversary, a telegram was received from President Roosevelt, following which official greetings from the Anatomical Society of Great Britain and Ireland were presented by Dr. John Beattie, Director of Research of the Royal College of Surgeons in London. Letters of felicitation, also, were read from every anatomical society in Europe. Incidentally, it was noted that the American society is one of the three oldest, having been organized less than a year after the British and German organizations. The rest of the evening was devoted to a scholarly presentation of the history of the American association by the retiring President, **Dr. Frederick T. Lewis,** of Harvard University. He divided the life span of the Society into three periods, the first of which, extending through the presidencies of Joseph Leidy, Harrison Allen, Thomas Dwight, Frank Baker, and Burt G. Wilder, was characterized by almost exclusive attention to gross anatomy as an adjunct of surgery. In the second period, under the influence of Huntington, Minot, and Mall, American anatomists entered upon active morphological research, participating especially in striking advances in the field of descriptive embryology. In the third period, initiated by Harrison's discovery of the method of tissue culture and continuing to the present time, the work of the Association has become predominantly experimental. Reminiscences of the first two periods were then given by former presidents of the Association, **Dr. J. P. McMurrich,** of Toronto, and **Dr. C. F. W. McClure,** of Princeton. Rising to a toast, **The Future of Anatomy,** former president **Herbert M. Evans,** of California, humorously remarked that, in consequence of the polygamous union of physiology with chemistry and physics, there had been delegated to anatomy the significant task of interpreting function in terms of structure.

The opening session, likewise striking a festival note, was given over to other past presidents of the Association: **R. G. Harrison,** of Yale, speaking on factors in the development of the ear; **R. R. Bensley,** of Chicago, on plasmosin, an important constituent of protoplasm; **C. M. Jackson,** of Minnesota, on the nature of the abnormally rapid increase in body weight following suppression of growth; **Florence R. Sabin,** of the Rockefeller Institute, on tuberculous allergy without infection; **G. L. Streeter,** of the Carnegie Institution, on the origin of gut endoderm in monkey embryos; and **C. R. Stockard,** of Cornell, on structural disharmony in the development of upper and lower jaws following extreme hybridization. The paper of **W. H. Lewis,** of Baltimore, illustrated by moving pictures of lymphocytes and monocytes in tissue cultures, was deferred until the last morning of the sessions.

On the second and third mornings, there were four round table discussions, all largely attended. The first, under the chairmanship of **Edgar Allen,** of Yale, tetempted to cover the entire endocrine control of the female sexual and reproductive functions in monkeys and other primates. The second, under **A. W. Meyer,** of Stanford University, discussed some aspects of the pelvis and vertebral column. The third, under **J. C. Hinsey,** of Cornell, dealt with visceral afferent pathways and visceral sensation. The fourth, under **Hal Downey,** of Minnesota, restricted itself to the lymphocyte. Abstracts of these discussions will appear in the May number of *The Anatomical Record* (**71**: No. 1, 1938).

In conjunction with the anthropologists, several anatomists participated in a symposium on growth and at a joint evening session in the Mellon Institute, **Dr. Franz Weidenreich,** of Peiping Union Medical College, presented his striking studies on the thirty-eight specimens of Sinanthropus Pekinensis—an ancient race interpreted as antedating the "ape-man" of Java. In a final joint session, six subjects were discussed: the fixation of the facial skeleton to the cranial base **(W. M. Cobb,** of Harvard University); rare features in the tibia **(Aleš Hrdlička,**

of Washington, D. C.); body build and pelvic type in white women (**W. W. Greulich**, of Yale); calcification patterns of human deciduous teeth (**I. Schour**, of Illinois); regional trends in human malformations (**W. F. Peterson**, of Illinois); and the pigments and color of living human skin (**E. A. Edwards**, of Harvard).

The Friday afternoon session was devoted to the viewing of sixty-eight demonstrations, 9 of which required moving pictures.

In the remaining three half days, 170 short papers of 10-minute length were reported, these being classified approximately as follows: endocrinology, 51; neurology, 41; embryology, 26; histology and blood, 26; gross and comparative anatomy, 16; miscellaneous, 7. Abstracts of these may be seen in the March 25 number of *The Anatomical Record* (**70**: No. 4, Suppl. 3).

Officers elected for the ensuing two years or more are as follows: President, **Stephen W. Ranson** (Northwestern University); First Vice-President, **T. Wingate Todd** (Western Reserve University); Second Vice-President, **Albert Kuntz** (St. Louis University); Secretary-Treasurer, **Eliot R. Clark** (University of Pennsylvania); Executive Committee, **George W. Corner** (University of Rochester) and **Olof Larsell** (University of Oregon).

Book Reviews

Thoracic Surgery. By Ferdinand Sauerbruch and Laurence O'Shaughnessy. Cloth. Pp. 394, with 215 illustrations, and 15 full page plates in color. Baltimore, 1938, Wm. Wood and Company. $13.50.

The volumes on thoracic surgery by Professor Sauerbruch have been admired and studied with care by thoracic surgeons everywhere. The appearance of this abridged "Sauerbruch" in English will be welcomed on every hand. The authors state in their preface that their chief aim in this volume is to reach an audience less directly concerned with the surgery of the thorax, viz.: "the general surgeon or physician, the tuberculosis specialist and more especially the general practitioner."

The entire subject of thoracic surgery is briefly reviewed in the pages of this monograph and the practices of the Sauerbruch clinic are described. A useful and up-to-date bibliography is appended containing a fairly large number of references to English and American contributions. The illustrations are excellent and the beautiful color plates add much to the book. One looks in vain through the text, however, for the name or names of the artists who have made so important a contribution to this work. The liberal use of illustrations by the authors indicates that they value highly the capacity of the eye to grasp situations which mere description cannot elucidate or define properly. It is not without interest that Andreas Vesalius in his *De Fabrica Humani Corporis* omitted to mention the name of his brilliant artist, Jan Stephan von Calcar, whose artistic creations added much to the "Fabrica"; yet posterity has rescued his memory from oblivion.

This reviewer was somewhat astonished to find the operation of posterior thoracoplasty described as being performed still from below upward. The reversal of this procedure by Alexander has met with such universal approbation by thoracic surgeons in this country that it seemed unlikely that the older sequence was still in use. In the light of the emphasis placed by the authors on the removal of the paravertebral segments of the ribs in thoracoplasty for tuberculosis, it is somewhat surprising to note in their illustrations (pages 190 and 192) how long the residual unresected paravertebral segments of the ribs are. In the treatment of empyema by intercostal drainage and suction, no mention is made of the importance of the admission of air to the pleural cavity or employment of irrigation to secure satisfactory drainage. In the recognition of wounds of the heart, the authors make no mention of the increased venous pressure and the use of the fluoroscope—the two agents of greatest value in detection of tamponade of the heart. The chapters on pulmonary suppuration, carcinoma of the lung, and malignancy of the esophagus are well done. The authors extol paraffin plombage for the treatment of certain types of pulmonary abscess. The historical aspects of each problem are related in sufficient detail to acquaint the reader properly with the advance in knowledgement and accomplishment of each subject under consideration. The authors direct attention to the fact that all paraesophageal (hiatus) hernias described by the roentgenologist will not be found by the surgeon and urge caution in operating upon these unless some viscus other than the stomach is engaged in the sac. The text concludes with an appendix in which O'Shaughnessy's operation of cardio-omentopexy for cardiac ischemia is described.

This abridged "Sauerbruch" should prove eminently useful and should find favor amongst a wide audience of American readers.

The Roentgenologist in Court. By Samuel Wright Donaldson. Cloth. Pp. 230. Springfield, Ill., 1937, Charles C. Thomas, Publisher. $4.00.

The author has presented the relationship of the physician and medical practice to the law in a manner which is authoritative and yet which makes enjoyable reading. The title is almost too exclusive considering the scope of the book. Although written expressly for the radiologist, it should be of interest to all practitioners of medicine.

It is unusual to review a book by citing the table of contents, but in this instance one can consider that each subject is adequately covered, and an index of its contents shows the wide interest it should have for all physicians.

Any physician may be called into court at any time to testify in a medicolegal proceeding. Because of this fact, the following chapters will be found to be of great interest: "Evidence and Testimony," "Privileged Communications," "Expert Testimony," "Expert Witness Fees," and "Doctor, Take the Stand."

Of importance to physicians in relation to their business arrnagements with each other and with their hospitals is the discussion of physicians and contracts.

Other subjects of general interest are: "The Relationship Between Physician and Patient," "Malpractice," "The Physician and the Law of Agency," and "Malpractice Defense and Prophylaxis."

Primarily of interest to the roentgenologist are the chapters on "X-ray Films as Evidence," "Ownership of X-ray Films," and the introduction which deals with the legal question as to whether the practice of roentgenology is the practice of medicine.

A Practice of Orthopaedic Surgery. By T. P. McMurray. Pp. 471, with 178 illustrations. Baltimore, 1938, William Wood & Co. $5.00.

The author presents a small but concise volume on the practice of orthopedic surgery. As such it is a worth-while addition to the library of the general practitioner and orthopedic surgeon, too. The general practitioner will find simply stated the various lesions commonly seen in orthopedic work and their more useful types of treatment. The orthopedic surgeon will find the viewpoint of an eminent orthopedic surgeon clearly expressed. The author's heritage is pre-eminent coming into the workshop of his predecessors, Hugh Owen Thomas and Sir Robert Jones, both of whom are usually regarded as the outstanding leaders of the medical profession toward the present methods used in orthopedic surgery.

One looking for minute detail of operative technique or treatment will be disappointed, for the detail is lacking. One looking for extensive discussion of controversial subjects will be disappointed because for the most part the author has given his views and left those of others alone.

The illustrations are perhaps too scanty, but for the most part they are clear and well chosen. The diagrams and drawings are a bit too simple perhaps, but again they fit in with the plan of the book. For one looking for a brief but concise treatise on orthopedics, the volume has much to recommend it.